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NOTE

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SPEECH DISORDERS

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THE ETIOLOGY OF STAMMERING; AN EXAMINATION INTO CERTAIN RECENT STUDIES; WITH A GLANCE INTO THE FUTURE

ELMER L. KENYON, A. B., M. D.
Associate Clinical Professor in Otolaryngology (Disorders of Voice and Speech) Emeritus, Rush Medical College, University of Chicago
Chicago, Illinois

I. Arnott, Bluemel, and Makuen

In introducing his own study of the etiology of stammering, in 1912, C. S. Bluemel, A. M., M. D., psychiatrist, stated the attitude of his predecessors on this subject in part as follows: [1]"We shall consider first the theory of its cause that finds at the present time the widest acceptance among pathologists. This theory supposes that stammering is due to a delay in vocalization; in other words, to the stammerer's inability to produce voice. The theory was first promulgated by Dr. Neil Arnott, in 1827, in his 'Elements of Physics.' Later the theory was supported by Merkel in Germany. Merkel no doubt advanced the theory independently, as many other writers have done since the time of Arnott."

Thus Arnott, Merkel, Wyllie, Bastian and others had conceived of stammering as due to some unknown manner of faulty "delay" or "hesitation" in the action of the laryngeal mechanism of voice production. While the conception was vague and incomplete it did locate the division of the speech function which primarily seemed to its sponsors to be at fault.

The attitude of the pre-Bluemel students as to the etiology of the disorder was, (1) to analyze the peripheral phenomena of stammering, and (2) to attempt to conceive of their cerebral explanation. In

[1]"Stammering and Cognate Defects of Speech," by C. S. Bluemel, Vol. I, page 181.

truth, however, little progress was actually made in either of these two basal aspects of the problem.

Dr. Bluemel, while still adhering to the belief of Arnott that the basal impediment in the speech was due to a default in the production of voice, attributed the defection in the production of voice not directly to failure in the action of the larynx, as had been conceived by the theory of Arnott, but indirectly by the "recoil" from the mind of the vowel succeeding the sound stammered on. The basis of this theory, in so far as it is related to the symptoms of the speech disorder, has now been definitely refuted.[2] The author (1) did not appreciate definitely what the psychophysiologic act of stammering was; (2) did not understand closely the psychophysiology of laryngeal action; (3) attempted to explain stammering only on the consonant and not on the vowel, which is just as important as stammering on the consonant; (4) failed to appreciate the importance of sound sequence in the word in determining the sound stammered on, and, finally, did not appreicate the absence of a definite distinction between the psychophysiologic character of vowel and consonant sufficient to render the "recoil" theory possible.

In a later book[3] than that already quoted, Dr. Bluemel elaborates his conception of the etiology of stammering; he now conceives of the speech disorder as an "inhibition," and employs the conditioned reflex conception of Pavlov in its explanation. This conception will be commented on later, in connection with the psychologic conceptions of other authors.

G. Hudson Makuen, M. D. (1855–1917), an otolaryngologist of wide understanding of medicine and speech, overlapped the period of Arnott and of Dr. Bluemel, and was sympathetic with both of their conceptions. At various times Makuen had made tentative attempts to connect the peripheral phenomena of the disorder with their possible cerebral cause.[4] In 1915 he regretted that a cause for "the faulty laryngeal action and consequent delayed vocalization of Wyllie could not be found." In the same essay, apparently influenced at least to some degree by Dr. Bluemel's then recent book,[5] and certainly having the aphasias in mind, he made what may turn out to be a significant statement, "Stammering probably has a physical basis which may or may not be demonstrable." In 1916, the year preceding his unfortunate sudden death, Makuen wrote,[5]"Stammering, at least in its chronic forms, is essentially a psychoneurosis, whatever may have been the original cause."

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[2]"A Critical Examination of the Foundations of the 'Recoil of the Vowel' Theory of the Cause of the Impediment of the Speech in Stammering," by Elmer L. Kenyon, M. D., *Journal of Speech Disorders*, Vol. V, No. 2, June, 1940.

[3]"Stammering and Allied Disorders," by C. S. Bluemel, M. A., M. D., New York, the Macmillan Company, 1935.

[4]"The Psychology of Stammering," by G. Hudson Makuen, *New York Medical Journal*, pp. 2 and 3, July 17, 1915.

[5]"Psychology of Diseases of the Respiratory Tract," by G. Hudson Makuen, M. D., *New York Medical Journal*, September 23, 1916, p. 12.

II. Psychologists and Psychiatrists

[6]In the Quarterly Journal of Speech for October, 1937, appears a more or less comprehensive statement of each theoretic conception of the etiology of stammering, by the following psychologists and psychiatrists. Each statement of theory is officially approved by its original sponsor. The statements appear in the following order:

Smiley Blanton, M. D., Psychiatrist, Cornell Medical College, New York City; Lee Edward Travis, Ph. D., Psychologist, University of Iowa (now of the University of Southern California); C. S. Bluemel, M. D., Psychiatrist, Denver, Colorado; John M. Fletcher, Ph. D., Psychologist, Tulane University, New Orleans, La.; James Sonnett Greene, M. D., Psychiatrist, Medical Director of the National Hospital for Speech Disorders, New York City; Mrs. Mabel Farrington Gifford, Chief of the Bureau of Speech Correction for the State of California. I also include in the list a [7]study of the etiology of stammering by Meyer Solomon, M. D., Psychiatrist, Northwestern University Medical School, Chicago.

I submit that this is a formidable array of psychologic and psychiatric talent for me, an otolaryngologist, to approach as a body critically, in the matter of their efforts to solve the etiology of stammering. But perhaps, unawares, they really do need to confer with me.

Each of these students presents some manner of individual abnormal psychology as the sole explanation of the disorder of stammering. The assumption that a general psychologic disorder, of whatsoever kind, can be the sole explanation of stammering may turn out to be correct or incorrect. After all it is an assumption. No proof has been offered. As to what particular kind of psychologic abnormality may be the cause of stammering the authors themselves are in disagreement.

Moreover, as it seems to me, the manner of their approach to the problem is unquestionably faulty. The very definition of stammering, as its fundamental idea, must conceive of a speech disorder of a peculiar and unusual type, involving complete stoppage of the speech; or of its definite, characteristic retardation, usually attended with struggle, while devoid of clean cut stoppage.

Succeeding Makuen, an otolaryngologist, these psychologic and psychiatric students have all diverged completely away from the conception of Arnott and his many followers, whose minds were primarily focused on the basal symptom of stammering, i. e., the impediment of speech, and who felt that this speech disorder must, primarily, as the fundamental symptom, be understood and explained. On the other hand, these later students have made no attempt whatsoever either to define, or even to closely study, these complex

[6]"A Compendium of Some Theories and Therapies of Stuttering," by Eugene F. Hahn, University of Southern California. Vol. XXIII, No. 3; Oct., 1937, p. 378.

[7]*The Journal of Speech Disorders*, December, 1939.

peripheral phenomena of speech, but have apparently jumped, without reasoning, at the conclusion that whatever the primary speech disorder might turn out to be—if some one should chance to closely study it—it must be what it is solely because some manner of general psychologic or psychiatric disorder serves to make it so. In view of the psycho-physiologic definiteness and complexity of this speech disorder, and in view of its etiologic significance as the basically most important symptom of stammering, this assumption that it must be based on general abnormal psychology, is not clearly founded, and is likely to turn out to be untrue.

To these authors, stammering is an emotional, or personality, or "cerebral dominance," or psycho-analytic, or other manner of abnormal psychologic disorder, each of which manner of psychologic or psychiatric disturbance must, according to its particular sponsor, as a matter of course, explain the impediment of speech, no matter what its nature may be. But if emotion, or disordered personality, or disturbed cerebral dominance, or psychoanalysis, or other psychologic disturbance, each of which conceptions some one, or more, of these authors upholds, could alone produce this strange characteristic stoppage and struggle in the action of the speech mechanism, *regularly in all cases*, we should be told exactly how; but no such explanation has been offered, or, evidently, since the speech phenomena have not been closely studied, even conceived. By failing to study closely the nature of the impediment of speech as the basal symptom, these students, it seems to me, have placed themselves in the anomalous position of seeking an explanation of a complex phenomenon the nature of which they have not even deemed it worth while to try to understand. Naturally such a narrow approach to the problem has attained to very little etiologic consequence. We are practically as far from attainment by such studies of a real solution of the etiology of stammering as when these studies were begun, more than a quarter of a century ago. Why? Because the fundamental symptom, the impediment of the speech, is just as far from solution as it ever was. Moreover, treatment of the disorder, leading *systematically* to recovery, or even to important improvement, is still, as every close student of stammering well knows, unattained by the psychologic or psychiatric methods evolved from such etiologic studies.

III. The Impediment of Speech

The reasons why I believe that stammering is primarily a speech disorder I shall begin to suggest in this essay, but I now wish, as a part of this immediate discussion, to state what, in my observation, the primary, basal, impediment of the speech really is on the peripheral side. This statement represents a close and persistent study of the peripheral facts of stammering. Since the speech machine necessarily can produce but one chest-laryngo-articulative-palatal act (on which each sound of speech depends) at the same time, each act of talking, as well as each impediment of the speech in stammering, must have reference to the production of a single sound. The present

statement will concern only the typical complete impediment of the speech, the stoppage, or "blocking," and not its various modifications and distortions that produce merely struggle and retardation.

When in the course of speech the stammerer finds himself unable to produce a certain sound the speech machine, nevertheless, continues in full action in each of the chest, laryngeal, articulative and palatal divisions. In spite of this continuation in the action of the muscles of speech, in all of its divisions, the *movement* of the chest wall and the passage of breath at the larynx, momentarily cease, and no vocal sound is forthcoming. Disregarding at this time the comparatively unimportant secondary muscular overaction, especially for articulation, the articulative and palatal activities are in principle produced normally.

During a complete and typical act of stammering, then, the physiologic status is that of the speech mechanism continuing in action for each of its divisions as if to produce speech, with articulative and palatal placements made in principal normally, and yet with complete absence of the passage of breath through the larynx and the production of voice. This manner of disturbed action of the speech mechanism in talking is not only identical in principle for all stammerers, which if caused by emotion, or "neuroticism" or other abnormal psychologic states, would in itself be most remarkable, but psychophysiologically it is far different from a mere "hesitation" in the speech. Only one explanation for such a psychophysiologic disordered action of the larynx is possible, namely, that the chest-laryngeal mechanism is acting not to produce, but to *prevent* the passage of breath and production of voice. All single acts of stammering, whether a stoppage, complete and typical, or a retardation, being a partial and distorted stoppage, are based on the above stated abnormal action of the chest-laryngeal muscular mechanism.

Such is the essential abnormal peripheral act of stammering, the close study of which recent students of etiology have completely disregarded.

I now venture to express my carefully considered opinion that the origin of stammering, complicated and extremely difficult of understanding though it be, nevertheless has no necessary causal relation to abnormal personality, to emotion, or to psycho-analysis or to disordered cerebral dominance, or to other general psychologic abnormality of behavior. And also I venture to express my fully considered opinion, that this extraordinary basal symptom of the speech disorder undoubtedly does demand clear psychophysiologic understanding, before the etiology of stammering can possibly be discovered.

IV. Three Possibilities

As a necessary basal step in the etiologic solution of the problem, the disorder of speech still cries out for understanding. That stammering might be *primarily* a speech disorder, having a psychophysiologic abnormality of action as its basis, which, by reason of the

compelled stoppage and retardation in the laryngeal production of voice coupled perhaps with other unrecognized psychologic influences, could be the *cause,* rather than the consequence, of the well known emotion and peculiar behavioristic social attitudes of the stammerer, seems not to have been conceived by these recent psychologic and psychiatric students. One of these two points of view is right and the other is wrong; either (1) stammering is basically and primarily a speech disorder, involving an essential and remarkable psycho-physiologic impediment in the production of speech, which, complicated by certain psycho-social influences, leads to the emotion and peculiar behavioristic attidutes of the stammerer; or (2) stammering is basically an emotional, or a personality, or a disturbed cerebral dominance, or a psycho-analytic or other manner of general psychologic disorder which, through some mysterious unknown chance, superinduces in every case these definite, closely similar, inexplicable, complicated, peculiar characteristic abnormal muscular activities of the speech mechanism during speech; or (3) as a compromise conception, stammering, although basically a speech disorder, might (perhaps only in certain instances) be influenced as to its origin by general abnormal congenital, psychologic or psychiatric factors.

V. Kenyon

Concluding this present comment on certain recent studies of the etiology of stammering, I desire to call attention to an essay published in 1930 by myself,[8] which followed (independently) in the footsteps of Arnott, in locating the cause of the impediment of speech in a definite wrong action of the larynx, consisting of the complete closure of the vocal cords, thus preventing voice production. Since the presentation of that essay a long patient continuation of this study has served to confirm those earlier tentative impressions, and that essay will be referred to in a later continuation of this study.

VI. Are All Stammerers Psychologically Abnormal?

Let us now inquire for a moment how certain of these recent students of the etiology of stammering may have arrived at the conclusion that all cases find their origin in "emotion," or "neuroticism," or other "abnormal personality" factors on the part of the stammerer. The authors themselves do not answer this query. Apparently they view the extraordinary speaking behavior of the mature stammerer, and note that certain individuals are definitely "neurotic," or extremely emotional, or possess other personality abnormalities, and conclude, therefore, since all stammerers behave in talking much the same, that all are, from a congenital standpoint, psychologically abnormal. But stammering *begins* in childhood, and

[8] "The Peripheral Physical Inhibition of Speech, an Essential Phenomenon and an Important Causal Factor of Stammering," Elmer L. Kenyon, M. D., *The Archives of Otolaryngology,* December, 1930, Vol. 12, pages 769–784.

the susceptibility to, and the repeated production of, disturbed speech with its consequent embarrassment, is certainly capable, as is commonly agreed, of producing subjective psychologic disturbance of a more or less serious degree, and, especially, and quite naturally, is capable of superinducing emotion in social relations even in the normal individual. Excepting as to the stammerer whose "neurotism" is congenitally extreme, the *only* time in which the congenital psychologic normality or abnormality of the stammerer, during the period of stammering, can be certainly determined is either before, or reasonably soon after, the disorder in talking has begun. But of this evident truth we hear nothing.

Moreover, I cannot help wondering whether the recovered stammerers of these students indicate definitely to them in all cases the kind of congenital psychologic abnormality that, because they stammered, has been attributed to them. I would like to have them point out the congenital psychologic abnormality that originally superinduced stammering in *my* recovered stammerers. I myself have failed to discover such abnormality.

My own point of view leads me to search not chiefly for *congenital* psychologic abnormalities, but for the *acquired* abnormal psychologic *effects* of the stammering. And these I have no trouble in detecting. They are indicated as acquired and not congenital largely by the fact that in case the stammerer recovers they are eradicable as and after the stammering ceases.

In support of my contention that stammerers are by no means necessarily abnormal psychologically in a congenital sense I now refer to other close students of stammering who hold the same belief; The senior Herman Gutzmann, M. D., late of Berlin, unquestionably much the greatest student of disorders of speech who has yet lived, and under whom I was privileged to be a pupil, told of a well known man of middle age in Germany who stammered severely, frequently, and constantly, and yet who was never emotional. No one who realizes the depth of Gutzmann's knowledge in this subject would dare to question his capability of observation in such a matter. At this time I will not appeal further to Gutzmann in this connection, as I may do at another time, but will state that I myself have observed long and closely an almost identically similar case of very severe and constant stammering in a young man just over twenty, evidencing little emotion. And, indeed, I believe that in all cases stammering occurs without, as well as with, emotion.

A second important student of disorders of speech, the late G. Hudson Makuen, M. D., of Philadelphia, under whom I was also privileged to be a pupil, in 1916, at the end of a review of the then current theories as to the etiology of stammering, made the following wise comments: [9]"Another neurologist claims that the defective inhibition found in stammerers may best be explained in terms of

[9]"Some Recent Theories on the Causation and Treatment of Stammering," G. Hudson Makuen, M. D., Philadelphia. *The Laryngoscope*, St. Louis, May, 1916.

the tics, and that stammerers are for the most part neurasthenic and psychasthenic, and it is well known that many stammerers belong to this class, although it is also well known that comparatively few neurasthenics and psychasthenics are stammerers, and many of those having various forms of tics show no disturbance of speech whatsoever. The problem of finding a causal factor which is common to all stammerers, therefore, still remains unsolved."

[10]Dr. C. S. Bluemel found the exhibition of extraordinary emotion not at the origin of stammering but only towards and at its maturity, where, as I have intimated, it quite fails to help out the theories of recent psychologic and psychiatric students.

Certainly, if, from a congenital standpoint, certain stammerers are psychologically normal and others are psychologically abnormal, no theory which applies to the latter class alone could possible serve, as Makuen intimates, and as all must agree, as an explanation of the etiology of the disorder. My own observation tells me that, excepting for the damaging influence on their minds and on their social behavior, of repeated stammering and of almost constant serious embarrassment, stammerers are for the most part as normal psychologically as any other average group of individuals.[11]

Another fact is worth consideration in this connection. Stammering not only injures the stammerer psychologically, but also psychophysiologically, as to voice production. My treatment of stammering regularly includes not only the attempt to eliminate the speech disturbance, and its psychologic effects, but also to correct the important and otherwise usually permanent characteristic psychophysiologic disturbance in the production of voice. Indeed I do not see how stammering can be systematically treated with success without especial consideration of this acquired wrong psychophysiologic habit of voice production, which I shall discuss at a later time.

Whether the congenitally psychologically normal or the psychologically abnormal class of children be under discussion, the *enormous majority* of individuals, as Makuen suggests, in either class, pass through the long years of speech development of childhood, when stammering is especially prone to develop, and on through the years of youth and adulthood, without any subjective or objective indication whatsoever of being in danger of developing stammering. I have watched especially the highly emotional individuals, who seem to be utterly free, excepting in rare instances, from danger in this direction. And the same is true of normal children. Only rarely is one caught in the "trap" of stammering. If this statement be true, no such direct causal relation on a mental basis alone between the psychologically, congenitally abnormal individual and stammering, as is conceived by recent theorists, is likely to be true, but, on the other hand, such facts inevitably must lead one to seek an especial

[10]The writer cannot now recall where in Dr. Bluemel's voluminous writings this statement appears.

[11]When the writer shall arrive at the discussion of the psychology of stammerings, in a future essay, he will be able to throw additional light on this subject.

causal factor to account of the *occasional* stammerer from either the congenitally psychologically normal or abnormal group.

If it be true, and it may be, that the psychologically abnormal child is relatively more susceptible to the danger of developing stammering than the normal child, the difference in degree of susceptibility seems to me, at this time, not to be highly conspicuous.

All of which discussion thoroughly discredits the idea that stammering is explicable on the basis solely of excessive emotion, or of "neuroticism," or of "abnormal personality."

VII. Why Recent Efforts Have Failed

Because of the many students who have conceived independently that stammering was basically a disorder of voice production the Arnott conception cannot be lightly cast aside. The failure of the Arnott School to attain to important etiologic results is hinted at quite directly by the above statement of Makuen, that a cause for "the faulty laryngeal action and consequent delayed vocalization of Wyllie could not be found." Why it could not be found may have been due to the failure to search for it beyond the merely superficial observation of stammering as a disturbance in the psycho-physiology of laryngeal action and of voice production. These investigators perhaps started right but stopped their investigation while it was still on its most superficial level.

To conceive of understanding the impediment of speech in stammering as a disturbance in voice production, without attempting to understand closely the psychophysiology of laryngeal action, constituted a vital desideratum on the part of these students. Only such psychophysiologic laryngeal studies could possibly put the real teeth into this theory which it may yet be shown to possess.

Stammering is fundamentally a disordered action of speech, or, perhaps, of voice production. This constitutes its basically determinative symptom. Any successful conception of etiology of the disorder *must*, at any rate, explain this symptom. For recent psychologists and psychiatrists to theorize that congenital psychologic, or psychiatric, disorders, or disturbances, could superinduce this complex disorder of speech, without even trying to understand closely what that speech disorder was, and to indicate definitely how their theories could explain it, was to work with a hopeless vagueness of conception. The psycho-analysts were able to perceive only the particular superficial aspects of the impediment of speech which fitted into the needs of their theory, and, therefore, they of course likewise fall into the general failure to solve the problem. Moreover, those authors whose theoretic conceptions are based on the universal congenital "neuroticism" or "personality disorder" of stammerers, in view of the existence of the strongest evidence that a very large proportion of stammerers are psychologically normal, have, as I believe, failed doubly to solve the problem. And since only occasionally does the psychologically normal, or abnormal class of children

suffer from stammering, it becomes absolutely necessary to explain why *only* these particular children, and not all in each group, are affected. As Makuen intimates, a real etiologic theory as to stammering demands an explanation of the speech or voice, disorder, as suffered by both the psychologically normal and abnormal child, and this, in so far as my knowledge goes, no one has as yet intelligently attempted.

Seeking the real reason for the recent failures on the part of psychologists and psychiatrists to attempt in a complete sense to solve the etiology of stammering, one should bear in mind that the function of speech is a psycho-physiologic function. The factor of physiology is present in almost all problems of functionally disordered speech, and rarely is a functional disorder of voice or speech completely soluble solely on a general psychologic, or psychiatric, basis. The trouble is that the field of study and work in the United States in disorders of voice and speech, speaking of the individual student, is top-heavy with a knowledge of general psychology and psychiatry, and is pathetically weak in the equally essential realm of specific psychophysiologic knowledge of voice and speech production; and so long as this one-sided status of individual knowledge exists among students of speech disorder, such unbalanced studies as those under discussion are, it seems to me, likely to continue.

VIII. The Future

After very many years, even centuries, of effort, by scores of students, the complete failure to solve so complex a problem as the etiology of stammering, strongly suggests that new significant knowledge on which the solution shall finally depend, must first be brought to light. The facts already understood do not offer a solution. Unless one finds as yet undiscovered patent facts, a solution will continue to remain impossible.

The most obvious, striking and significant symptom of the disorder, its onset during the speech development period of childhood, has curiously been given little attention by students of etiology. Approximately ninety-five per cent of the cases begin before the seventh year of age and most of these cases in the second, third, and fourth years. Nearly all instances of onset have ceased at fourteen years. The fact that stammering is a disorder in the production of speech and that it originates in most instances during the speech development period of childhood, certainly suggests that at any rate the impediment of the speech involved in stammering could possibly be related to difficulties in speech development. But, up to this time no one has deemed it worth while, for example, to delve into the psychophysiologic foundations, especially of the development of voice for speech purposes by the child. If the etiology of stammering is ever to be discovered, the effort to be made must, in my opinion, begin at the beginning, by acquiring a knowledge of how psychophysiologically the impediment of the speech, or perhaps of voice, is produced, and the significance of such impediment, a study which,

to my knowledge has never been seriously attempted. In order to solve this basal problem as to the nature of the speech impediment, I believe the following items of knwoledge must be acquired: (1) Definitely what happens psychophysiologically in the production of the impediment of the speech when the individual stammers; (2) the psychophysiology of laryngeal action in all its phases, including animal functions, as well as the production of voice; (3) the psychophysiology of voice development for speech by the child, and the possible dangers involved in that effort. With such knowledge in hand, coupled with the host of other facts generally understood, it may become possible to evolve an etiological understanding of the many questions involved in the problem somewhat as a novelist evolves the coherent details of his story.

IX. Summary and Conclusions

1. The purpose of this essay has been to clarify the present status of the study of the etiology of stammering, preparatory to presenting an entirely new etiological conception.

2. The author has recently shown the untruth of the "recoil" conception as to the cause of the impediment of speech in stammering.

3. The theory of Arnott, that stammering results from a faulty action of the larynx in producing voice, may be of real importance, but as yet it has never been completely worked out.

4. The author shows the incompetence of those recent theories that find the explanation of stammering solely in the congenital general psychologic abnormality of the stammerer, or in psychoanalysis, or in disturbed cerebral dominance, or in other psychologic abnormality. None of these theorists has deemed it necessary to understand closely either the basal symptom of the disorder, the impediment of speech, or its relation to the psychophysiology of voice and speech production, or to connect their theories causally with the impediment of speech. These failures render the theories under discussion unquestionably incompetent.

5. Evidence is presented indicating the congenitally psychologic normality of a very large proportion of stammerers, and this evidence strongly opposes the conception that the universal explanation of stammering lies in "neuroticism," or "disorders of personality."

6. To hope to understand the etiology of stammering without a close knowledge of the psychophysiologic origin and nature of the impediment of the speech, is, in the author's opinion, unthinkable, and this knowledge remains as yet undiscovered.

7. The author regretfully points out that the individual educational status of students and workers in the field of disorders of speech in the United States is at present top-heavy on the side of general psychologic and psychiatric knowledge, and is pathetically weak on the side of the specific psychophysiology of voice and speech production.

8. The author's own belief is that the impediment of speech is not a result of general psychologic or psychiatric disturbance, but

that it is caused by a specific psychophysiologic disordered action of the larynx in producing voice. He also believes that the characteristic behavioristic attitudes of the stammerer are caused by the embarrassment brought about in large part through the constant and serious uncertainty of his ability to talk normally. This conception reverses the order of cause and effect as presented by recent psychologic and psychiatric theories.

9. Indicating with apparent certainty the correctness of the author's etiologic conception, the employment of a new method of treatment based upon it has been attended with no failure to bring about complete and permanent eradication of the disorder at certain ages—nine, ten, eleven and twelve—during the past six years. Regularity of success in treatment, which indicates the practical possibility of the eradication of the disorder rationally after age twelve) thus has for the first time been achieved.

10. In an essay soon to follow the present, the author proposes to present a careful study of laryngeal action in its relation to the cause of the impediment of the speech in stammering.

THE PERSONALITY STRUCTURE OF STUTTERING. By J. M. Bender. Pitman Publishing Co., N. Y. 1939. 189 pp.

This is a useful handbook for those who deal with stuttering children or adults. Beginning with a review of literature on stuttering, its frequency, distribution and importance educationally, the author then discusses the nature, causes and treatment of stuttering (stammering). He gives an interesting treatment of the psychoanalytic point of view with an exposition of various methods used for the analysis of the stutterer's personality. There is a good digest of recent experimental work on the physiology of stuttering, and psychological significance of various findings. The author finds that stutterers tend to be above average, or at least average, in intelligence. Many are superior. He finds that more left-handedness and ambidexterity exist among stutterers than among normal speakers. Considerable space is devoted to an analysis of various Personality Schedules. This is followed by an intensive study of the personality characteristics of stutterers as compared with outside control groups. The final summary attempts to outline the differences between stutterers and non-stutterers, physically, mentally and emotionally. In some cases there is wide variance between the non-stutterer and the stutterer in any or all of these fields. In others the "margin of safety" is very narrow and near-normal or apparently normal, depending upon whether the stuttering is a post-pubertal or pre-pubertal development. The importance of early corrective measures is stressed. The book closes with a glossary of technical terms, an extensive bibliography and a good index. This book should be in the hands of every speech correctionist and candidate for a higher degree in speech pathology.

Sara Stinchfield-Hawk, Ph. D.,
Univ. Coll., Univ. of So. Calif.

THE RATE OF DIADOCHOCINETIC MOVEMENT OF THE JAW AT THE AGES FROM SEVEN TO MATURITY

RUSSELL L. JENKINS
Director of Speech
Isaac C. Elston Senior High School
Michigan City, Indiana

The production of speech demands the use of muscles, groups of muscles and organs the basic functions of which are not to produce speech. The overlaid function—the production of articulated speech—makes certain demands that the individual system, even though biologically and physically normal, may not be able to meet. The production of articulate speech demands manipulative movements of the jaw, lips and tongue that are much faster than those demanded by the basic functions of chewing, sucking and swallowing. For this discussion we are considering the reciprocal innervation of muscles that permit rapid movements of the tongue, jaw and lips.

The rate of diadochocinesis, (diadochocinesis is the making of repetitive acts by muscles, such as tapping the foot, tapping the finger, raising and lowering the brows, opening and closing the jaws) can be taken as an indication of the speed of change from inhibition to stimulation of antagonistic sets of muscles. This speed of change may be called the speed of the reciprocating synapses of the central nervous system. A test of diadochocinesis of the articulators is a measure of the maximum rate at which the reciprocating synopses of the central nervous system may function for speech uses. Most speech needs do not tax to the full the reciprocating synapses of the normal adult. There is usually a distinct margin of reserve speed. Occasional words, however, if spoken rapidly may approach the limits of diadochocinesis. The phrase "Methodist Episcopal Church," for example, may require movements of the tip of the tongue so fast as to be close to the limit of speech.

Diadochocinesis normally increases during childhood. Hence one of the determiners of the rate of diadochocinesis is the degree of maturation. Disease of the central nervous system, however, apparently slows up the rate of diadochocinesis. Some disturbances in the central nervous system affect the movements of the articulators in such a way as to cause the individual to produce a type of speech very much like stuttering. When a disturbance of the central nervous system causes stuttering speech, general sluggishness of the muscles of the face and mouth and other muscles which are involved in the production of speech is noted. Dr. Robert West's (Wisconsin) experimentation is illustrative of some results of certain disorders of the central nervous system. He noted that a disorder resembling

dysphemia, the overt manifestation of which is stuttering, followed a luetic lesion. This disorder was characterized by general sluggishness of sets of muscles controlling the raising and lowering of the brows, the opening and closing of the jaws, and the tapping of the finger. This sluggishness of muscles disappeared after a cure of the lesion. These observations seemed to indicate that there is a close connection

TABLE I

CORRECTION OF MALE RATES BASED ON FASTER MATURITY RATE OF FEMALES

Age	Males	Age	Females
7	3.68	7 (3)	3.84
8	3.93	8	3.90
9	4.18	9	4.13
10	4.22	10	4.23
11	4.78	11	4.32
	Pettit (2)		
14	5.00	12	4.70
15	5.20	13	4.80
16	5.40	14	5.10
17	5.60	15	5.30

TABLE II

ALPHA WAVE FREQUENCY AND RATES OF DIADOCHOCENESIS

Age	Average Alpha Wave Frequency (1)	Jaw Movement* Male	Female	Range of Alpha Frequency (1)
7 years	9.00	3.67	3.84	7.00–11.00
8 years	10.30	3.88	3.90	9.00–12.50
9 years	9.40	4.19	4.13	8.50–11.00
10 years	11.20	4.20	4.23	9.00–14.00
11 years	10.80	4.55	4.32	8.50–13.00
12 years	10.90	4.70	4.65	8.50–13.00
13 years	10.30	4.90	4.80	9.00–11.00
14 years	10.50	5.00	5.10	9.50–11.00
15 years	10.30	5.20	5.30	9.50–12.00
Adults	10.40	5.43	5.50	8.00–12.00

(1) Lindsey, p. 354.

*Pettit (2), Schlanger (4), This Thesis (3).

between certain types of disorders of the central nervous system and dysphemia. In order to determine whether this observation was significant in diagnosing stuttering patients it was necessary to discover whether there were non-pathological, significant differences between stutterers and non-stutterers in the speed of performing diadochocinetic movements. The objective of Dr. West's (5) experimental work was to discover the differences between stutterers and non-stutterers in diadochocinetic movements. He chose 64 cases of

adults from which to obtain statistics to show the maximum speed which stutterers and non-stutterers could attain in making diadochocinetic movements, opening and closing the jaws, raising and lowering the brows, and tapping the finger. The number of times the individual could open and close his jaw per second was added to the number of times he could raise and lower his brow per second in

TABLE III (5)

(1) Combined rate of movement of the jaw and brow.
(2) Hand-tapping rate.
(Rating of sixty-four subjects.)

No. of Cases	(1) Means	(1) P. E. of Means	(2) Means	(2) P. E. of Means
21 males, normal.....	9.26	.262	7.98	.159
18 males, stuttering.....	7.50	.172	6.93	.126
18 females, normal.....	8.15	.148	7.21	.880
7 females, stuttering...	5.98	.295	7.53	.258

Note: The hand-tapping rate was taken to check the technique by comparison with other studies.

TABLE IV (5)

DIFFERENCES IN THE JAW-BROW TEST

Between	Difference	P. E. of Difference	D./P. E.	Probability of Real Difference
Normal and stuttering males...	1.76	.313	5.62	9999.2 in 10,000
Normal and stuttering females..	2.17	.33	6.58	10,000 in 10,000
Normal males and normal females.....	1.11	.30	3.70	9,937 in 10,000
Stuttering males and stuttering females.....	1.52	.341	4.45	9,987 in 10,000

order to get an objective figure for comparisons. The results of the West study on combined movements of the jaw and brow were as follows:

1. The 21 normal males showed a greater spread of variation than the 18 normal females.
2. The normal males were 1.11 units faster than the normal females.
3. The normal males were 1.76 units faster than the stuttering females.
4. The 18 normal females were 2.17 units faster than the 7 stuttering females. The difference between the normal and the stuttering females is within .41 units of the difference between the normal and stuttering males.

The statistical results showed sufficiently small probable errors to suggest reliability of the results. In comparing the statistical results, we find important overlappings of rates for these diadochocinetic movements made by the stuttering and the non-stuttering groups. Out of the 39 stutterers, 11 overlapped the normals in their rates, and this overlapping occurred in the lower quartile of the normals and the upper quartile of the stutterers. The figures for the females showed that the overlapping was such that none of the stutterers' rates fell within the range of the middle 50 per cent of the normal group. These observations seem to indicate that a slow rate of diadochocinetic movement of the jaw and brow

> " . . . is either a cause of or is related to a cause of dysphemia." (5)

These results of West seem to indicate that the diagnosis of a speech disorder in any given case, especially if dysphemia is thought to be present, should include a neurological test based on diadochocinetic movements. Such a test might reveal a neurological weakness which might be the primary cause for the disorder. However, the disorder might be one of neuropathology and poor emotional and social adjustment, or it might be a case of neuropathology and metabolic disturbances, or it might be that there are other factors in combination with the neuropathology to cause the disorder.

Thus far experimental evidence seems to show a connection between dysphemia and the functioning of the central nervous system, as is shown by diadochocinetic movements. Following these results, West concluded:

> "It seems, therefore, as though there was another factor in the etiology of dysphemia in addition to . . . psychogenic conditions . . . another factor that may or must combine with . . . emotional habits to produce dysphemia." (5)

When it is established that a slow rate of diadochocinesis "is a cause of or related to a cause of dysphemia," a question of treatment arises. Will practice of diadochocinetic movements increase the speed of such movements and thereby be good therapeutic practice? Dr. Ream (3) found upon experimentation that practice of diadochocinetic movements enabled the individual to maintain better rhythm in performing the act, to produce more uniform movements throughout a period of testing, and to produce greater excursion in movement, but concluded that

> " . . . the motor neural set, undoubtedly an important condition of such basic ability is inherited." (3)

Having determined that rates of diadochocinetic movements are determined by an inherited motor neural set, one can set up norms for rates of certain diadochocinetic movements by using a sufficient number of normal speakers. Norms worked out for each sex and for each age would provide objective measurements to be used in diag-

nostic procedures. The fact that the rate of diadochocinesis varies with physical maturity and with sex, the fact that the greatest incidence of stuttering is for the boys, and the fact that the rate at some ages shows no increase over the rate for the previous ages make it necessary to establish norms for specific diadochocinetic movements for each sex and each age.

Bernard Schlanger (1) began the work of setting up a table of norms for the diadochocinetic movement of opening and closing the jaw. He chose his subjects from Lafayette, Indiana, Schools and established norms for the opening and closing the jaw at maximum speed for both males and females ages 7, 8 and 9. Schlanger's work is recorded in Table V.

Calvin Pettit (2) followed up Schlanger's work and established norms for the same movement for the two sexes at ages 12–17 inclusive. He chose his subjects from the Madison, Wisconsin, City Schools, the Princeton, Indiana, Schools, and the University of Wisconsin speech classes. His results are found in Table V.

The purposes of this research were to complete the table of norms started by Schlanger and Pettit and to inculcate into one work the work done thus far in the University of Wisconsin. The author used students from Emerson School, Madison, Wisconsin, and set up norms for the two sexes at ages 10 and 11. Norms for the ages 18 and 19 were established by using English and Speech Students of the University of Wisconsin. The correlation between the rates of diadochocinesis and age in months for 18 and 19 year old students was almost zero, so it seemed wise to assume that the rate matured at about the age of 18. The author then used University of Wisconsin students above the age of 19 years and 6 months to establish a mature rate, a rate for adults.

Table No. V is a composite table which gives the statistical results of all the work done thus far at the University of Wisconsin on this phase of diadochocinesis.

Interpretations:

Table V contains the norms set up by the three Wisconsin studies and are usable for comparative purposes in diagnostic procedure within the limits of their probable errors as indicated within the experimental limits of these studies.

Pettit's figures show that the females, ages 12–17, had a faster rate for this movement than did the males, except at ages 12 and 13. As females develop and mature more quickly physically than do the males, and as at the age of puberty, about the age of 12 for females, the females are considered to be two years more mature than the males of the same age, and as the speed for this movement has maturation rate, he (Pettit) compared the rates of males, ages 14, 15, 16, 17 with the rates of females ages 12, 13, 14, 15 respectively. Table I shows his results.

Considering that females are two years more mature physically than the males at the age of 12, that the females and the males are

COMPOSITE

No. of Cases		Age	Sex	Rate	P. E. of Mean	P. E. of Distribution	Age Difference of Same Sex Difference	P. E. D.	Probability (1)	Sex Difference–Same Age Difference	P. E. D	Probability
20		7	M	3.67	.145*	.66						
17			F	3.84	.680*	.165				.17	.22*	78
18	3	8	M	3.78	.640*	.151	.11	.16*	75			
21			F	3.90	.380*	.083	.06	.18*	63	.12	.17*	75
13		9	M	4.17	.710*	.197	.39	.25*	94	.04	.25*	56
20			F	4.13	.770*	.161	.23	.18*	90			
19		10	M	4.20	.084	.540						
27	5		F	4.23	.075	.574				.029	.112	57
30		11	M	4.55	.074	.598	.36	.111	98	.239	.098	57
29			F	4.32	.065	.518	.09	.075	73			
		12	M	4.70	.058							
			F	4.65	.053							
		13	M	4.90	.075		.20	.094	92			
			F	4.80	.059		.10	.080	79			
		14	M	5.00	.066		.10	.100	75			
	4		F	5.10	.073		.30	.094	98			
		15	M	5.20	.084		.20	.107	89			
			F	5.30	.070		.20	.102	90			
		16	M	5.40	.088		.20	.122	86			
			F	5.45	.054		.20	.890	93			
		17	M	5.60	.067		.20	.113	89			
			F	5.65	.052		.20	.075	96			
22		18	M	5.38	.098	.683	.13	.141	73			
21			F	4.94	.078	.507				.44	.123	99
20	5	19	M	5.25	.102	.677				.06	.127	62
16			F	5.31	.075	.446	.37	.107	99	.06	.127	62
21		20 &	M	5.43	.122	.830						
19		Over	F	5.50	.124	.800				.07	.17	57

*Standard Error instead of probable error.
(1) Probability in 100 cases.
(2) Figures for all ages in the *groups* indicated.

equally and similarly developed at the age of 6, and that in each succeeding year the sex differential accelerates, the author made changes in male rates by means of the geometric progression: ⅜, ¾, ½, 3, 6, 12. This assumption means that, during the 12 month period between the ages of 6 and 7, the girls' physical development is as much greater than the physical development of the males as ⅜ of a month more growth would allow. During the 12 month period between the ages 7 and 8 the females were considered as developing as much more than the males as ¾ of a month would allow. During the 12 month period between the ages 8 and 9, the females' physcial development was considered as being as much greater than the males' as 1½ months would allow. During the 12 month period

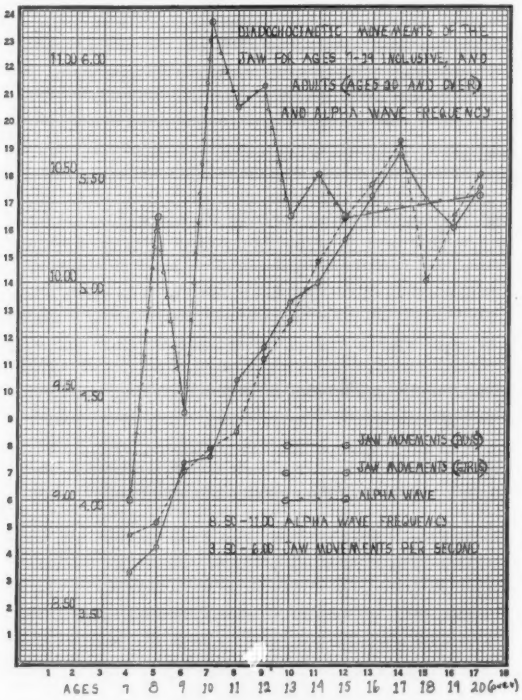
TABLE No. V

All Sex Figures (2)								Correlations				Means for Jaw-Movements Calibrated for Age and Sex Making Use of Probable Errors		
Males Mean Rate	P.E.M	Females Mean Rate	P.E.M	Sex Difference	P.E.D	Difference Probability(1)		Males r	P. E.r	Females r	P.E.r		Age	Sex
												3.5–3.8*.....	7	M
												3.7–4.0*.....		F
3.89	.11*	3.98	3	.08*	.09	.136*	75	.33	.12*	.21	.13*	3.6–3.9*.....	8	M
												3.8–4.0*.....		F
												4.0–4.4*.....	9	M
												4.0–4.3*.....		F
												4.1–4.3	10	M
4.42	.06	4.27	5	.05	.14	.08	87	.32	.09	.13	.09	4.2–4.3		F
												4.5–4.6	11	M
												4.3–4.4		F
												4.6–4.8	12	M
												4.6–4.7		F
												4.8–5.0	13	M
												4.7–4.9		F
												4.9–5.1	14	M
5.10	.333	5.20	4	.030	.10	.45	93	.443	.038	.558	.32	5.0–5.2		F
												5.1–5.3	15	M
												5.2–5.4		F
												5.3–5.5	16	M
												5.4–5.5		F
												5.5–5.7	17	M
												5.6–5.7		F
												5.3–5.5	18	M
												4.9–5.0		F
5.32	.07	5.13	5	.050	.19	.08	95	.015	.104	.083	.110	5.1–5.4	19	M
												5.2–5.4		F
												5.3–5.6	20	M
								.095	.15	.086	.15	5.4–5.6..and over		F

3 Figures for ages 7, 8, and 9, Schlanger, (4).
4 Figures for ages 12–17, Pettit, (2).
5 Figures for ages 10–11, 18–19, 20 and over, this thesis.

between the ages 9 and 10, the females' physical development was considered as being as much greater than the males' as 3 months would allow. During the 12 month period between the ages 10 and 11, the females' physical development was considered as being as much greater than the males' development as 6 months would allow. Thus for the 12 month period between the ages 11 and 12, the females' development would be as much greater than the males' development as 12 months would allow. Table I shows the male rates increased according to the given geometric progression. $\frac{3}{8}$ of the difference between the male and female rates for the age of seven was added to the established male mean for the age of 7; $\frac{3}{4}$ of the difference between the male rates for the ages 7 and 8 was added to the established male

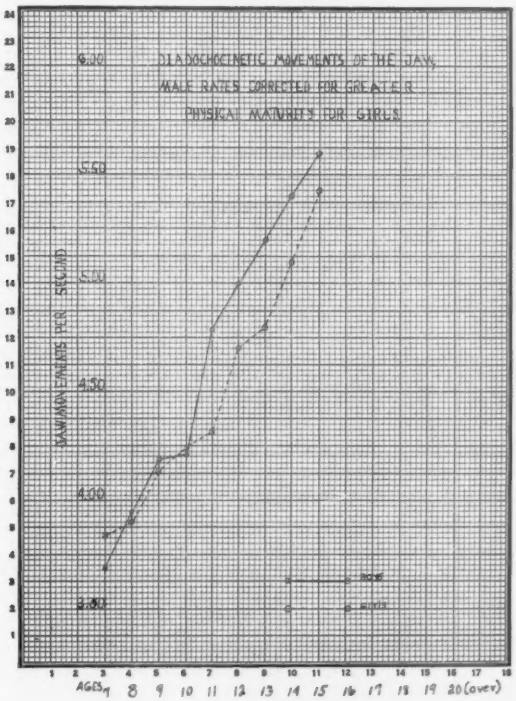
mean for the age of 8, etc. Table I shows these results and indicates that after this correction of male rates, the male rate exceeds the female rates for all ages 7–17 inclusive, except for the ages 7 and 10. Few male cases age 7 were used and the probable error was quite large; likewise, the probable error in the case of males age 10 was large, therefore, the accuracy of the figures for these two ages is less than that for the other ages.


DIADOCHOKINETIC MOVEMENTS OF THE
JAW FOR AGES 7-20 INCLUSIVE, AND
ADULTS (AGES 40 AND OVER)
AND ALPHA WAVE FREQUENCY
11.00 6.00
10.50 5.50
10.00 5.00
9.50 4.50
9.00 4.00
8.50 3.50
JAW MOVEMENTS (BOYS)
JAW MOVEMENTS (GIRLS)
ALPHA WAVE
8.50 - 11.00 ALPHA WAVE FREQUENCY
3.50 - 6.00 JAW MOVEMENTS PER SECOND
AGES 7 8 9 10 11 12 13 14 15 16 17 18 19 20(over)

Donald B. Lindsley, Western Reserve University, investigated spontaneous electrical activity in children and adults. These electrical vibrations were recorded over the occiputal region posterior to the division line between the parietal and occiputal lobes. Lindsley records only alpha wave frequencies, the alpha wave having a frequency of 20 vibrations per second or showing a 10 cycle rhythm. He reports that these rhythmical waves have a fairly regular rhythm when the child attains the age of about 3 months, and suggests that there is no satisfactory explanation of their onset at 3 months nor

their increase in frequency, but it does suggest a developmental process, and the rise in frequency noted in his subjects for the ages 10–12 suggests a relation to some of the many physiological changes at this time.

Table II shows Lindsley's alpha wave frequencies for the different ages and also the established norms of diadochocinesis of the jaw for the same ages. Comparisons in these rates and the graphic comparisons seem to indicate a relationship between the rise and fall of the



frequency of the alpha wave and the variation in diadochocinesis between the two sexes. As there seems to be some correlation between the alpha wave frequency and the rate of diadochocinesis, and, as Lindsley's figures show the adult frequency of females to be 10.4 and that of the males 10.8, we might assume that there is a difference in the rate of diadochocinesis between the sexes based on the neurological systems and not based on general development. This may suggest further study of the alpha wave and diadochocinesis.

Conclusions:

1. There seems to be an increase in the rate of diadochocinesis of the jaw from the age of 7 to the age of 18.

2. Statistically speaking, the rate for this diadochocinetic movement does not closely correlate with age, and the correlation shown is unreliable. However, these correlations were figured on three different groups: Schlanger's group with 89 cases gave one correlation; Pettit's group with 419 cases gave another correlation; and the author's group with 105 cases gave another correlation. Pettit's correlation with 419 cases was much higher than either of the other two correlations, as Table V shows, and the probable error of the coefficient of correlation is much lower than those of the other two groups. The small number of cases used in these studies doubtless was largely responsible for the low coefficients of correlation. However, in each group the trend is toward a significantly high correlation, and, had all the groups been taken together in the figuring of one correlation, the correlation would probably have been considerably higher and statistically more reliable.

3. There seems to be greater probability that there is a difference in speed of this movement for successive ages than there is that there is a difference in speed between the sexes of the same age.

4. There seems to be no correlation with age after the age of 17.

5. The female norm established here for adults is greater than the male norm for this diadochocinetic movement.

6. Male rates, corrected to compensate for greater female development at the various ages, seem to exceed the female rates.

7. The speed of diadochocinesis of the jaw seems to vary during the different hours of the day, the maximum rate being at 4 P. M.

8. The individual male rates seem to vary more widely from the established male mean than do the individual female rates vary from the established female mean.

9. There seems to be some correlation between the alpha wave frequency and the rate of diadochocinesis of the jaw.

10. Practically all subjects seem to produce their maximum rate for this diadochocinetic movement during the first 10 seconds.

11. Stutterers seem to show a slower rate of diadochocinesis of the jaw than do mon-stutterers.

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A STUDY OF BLOOD GROUP DISTRIBUTION AMONG STUTTERERS

JEANETTE ANDERSON AND MARY L. WHEALDON,
From the Laboratories of the University of Wisconsin

Despite the many studies that have been undertaken in the name of stuttering, we are still far from having to worry about Voltaire's injunction that "The secret of being tiresome is to tell everything." We have all too little to tell. We have found but few pieces which fit into the jig-saw picture which "at longest last" must give us at least a clue to the etiology of this outward manifestation of an underlying disorder.

It is our purpose to determine whether or not there is any relation between stuttering, this outward manifestation of an inner disturbance, and certain protein characteristics which control the agglutination of red blood corpuscles. In 1900 Dr. Karl Landsteiner discovered, while working in Vienna, that when red blood cells of one person are mixed with blood serum of another individual, agglutination or a clumping together of red cells sometimes occurs. Such agglutinations had occurred in transfusions and resulted almost always in the death of the patient because the red cells clumped together and could not pass through the blood vessels. In red blood cells Landsteiner found two specific protein substances called antigens or agglutinogens. It has since been shown that these red cell proteins are usually present at birth, are always established within the first few months of life and remain constant throughout life. Red cells may have the property of either, both or neither of these two protein substances that have been designated A and B. Thus, we have four blood types: A, B, AB and O. Correspondingly, the blood serum of an individual will contain antibodies opposite to the cell antigens. (i. e., if cells are A, serum will be β.) This reciprocity guards against auto-agglutination or the clumping together of red cells within an individual's body. It is, of course, equally important to guard against the clumping of cells in transfusion, and so we hear a great deal about "matching" the bloods of patient and donor. The four blood groups are inherited on the basis of Mendelian multiple allelomorphs.[1-2]

We want to know if the distribution of the four blood groups, A, B, AB, and O, is the same among stuttering individuals as it is in the general population. We are not attempting to establish a casual relationship between these factors nor are we trying to show genic connections between them. Our investigation is concerned only with blood group distribution among stutterers compared with that distribution in a large sampling of the general population.

[1]Snyder, Laurence H. Blood Grouping in Relation to Clinical and Legal Medicine. Baltimore. 1929.

[2]Snyder, Laurence H. The Principles of Heredity, pp. 93-100. Heath. N. Y. 1935.

So far as it can be learned, no study of these two factors has been made. As blood groups are determined by the protein agglutinins and agglutinogens of the blood, any significant deviation from the normal distribution of these groups might indicate a real constitutional difference between stutterers and non-stutterers.[3]

Procedure

Names of stutterers were secured from the records of the University of Wisconsin speech clinic and from the Rockford, Illinois, public school system. Fifty of these subjects were available for interviews and blood grouping. No matched control group was used because we were interested in determining only one relationship: whether or not there is an abnormal distribution of blood groups among stutterers. As our control group, therefore, we are using the 38,000 individuals[4] who have been grouped by other investigators in the United States as reported by Wiener.[5]

Since blood groups remain constant throughout the life of the individual and exhibit an equal sex ratio, and since about 99 per cent of a sampling from the general population would be non-stutterers, we feel that these earlier findings constitute an adequate control on our study. The fact that there is a 1 : 1 sex ratio of all types of blood groups would lead one to predict that stuttering would not classify itself as a correlate of any one blood group since stuttering has a high sex ratio, but, nevertheless, it seems wise to extend the study of blood groups to stuttering. Our method of grouping was the usual hospital laboratory technique employed at Wisconsin General Hospital. Fortunately, we were able to make our own groupings.

Findings and Discussion

Every individual exhibits certain unmistakable inherited characteristics. We are able to recognize and to classify these phenotypes. Eye color, hair color and pigmentation of the skin are some of the more obvious inherited characteristics. Another is this peculiar protein property of red blood cells that controls agglutination of them. Because this blood characteristic can be recognized and tested for, we were able to make this study.

Our control group includes the 38,000 individuals who have been typed in investigations carried on in the United States. The available findings on blood group distribution in the general population are:[6]

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[3]It is obvious that a large sampling of the general population must include a good many stutterers—at least 1% of the total number. We thus have a large sampling of non-stutterers mixed with some stutterers.

[4]As we noted before, probably 1% of this number is stutterers. That means that we have as controls about 37,620 non-stutterers mixed with 380 stutterers.

[5]Weiner, Alexander S. Blood Groups and Blood Transfusions, p. 158. Charles C. Thomas. Springfield (Ill.). 1935.

[6]Weiner, Alexander S., *op. cit.*, p. 158.

In attempting to correlate such factors as left-handedness and neurosis, for example, we would need a very large sampling of each group since we could never be sure of the exact classification of any given case. The sampling would need to be large enough so that those right-handers who were erroneously classified as left-handers would be "cancelled out" by those normals who were erroneously called neurotics. But in correlating sex and tuberculosis, on the other hand, fewer cases would be necessary, since each of the compared classes would have few, if any, aliens included. If twenty tuberculosis individuals were selected at random from the records of the general hospital, and it was found that nineteen of the twenty were female, a strong presumption of a correlation arises. In attempting to correlate stuttering and blood groups, we consider our

TABLE I

Location	Investigators	No. Persons	Per Cent of Groups O	A	B	AB
United States	Snyder	20,000	45.0	41.0	10.0	4.0
New York City	Tiber	10,000	45.6	36.4	13.5	4.5
Detroit, Mich.	Culpepper and Ableson	5,000	44.5	36.1	14.3	5.2
Rochester, Minn.	Sandford	3,000	44.5	42.3	8.7	4.5
Average		(total) 38,000	44.9	38.9	11.6	4.5

Our findings among this group of fifty stutterers were:

TABLE II

Group	No. Persons	Per Cent of Groups O	A	B	AB
Stutterers	50	46.0	44.0	4.0	6.0
Controls (average)	38,000	44.9	38.9	11.6	4.5
Range in U. S. Studies		44.5–45.6	36.1–42.3	8.7–14.3	4.0–5.2

group of fifty stutterers adequate because we are reasonably certain that all of the subjects stutter, and we can determine a definite blood group for each of them; both factors that we wish to test appear in each of our subjects. We need "cancel out" none of them.

What techniques can we apply to determine whether or not our group is large enough on which to base a conclusion and whether or not our finding is a significant one? When the test of chance halves of distribution among our fifty subjects, the consideration of the group in two equal and unselected sections, is applied, the distribution is essentially the same as it was for the entire group of stutterers:

This indicates that the sampling is large enough on which to base a conclusion. If we were testing a characteristic less universally present than blood groups, it is obvious that we should have to

consider a larger number of subjects in order to obtain fifty individuals exhibiting both factors we wish to test.

To determine the validity of our finding we can apply Garrett's formula for determining the reliability of difference in percentages.[7]

TABLE III

Group	Percentage Findings O	A	B	AB
Total group of fifty	46.0	44.0	4.0	6.0
First chance half	48.0	44.0	0.0	8.0
Second chance half	44.0	44.0	8.0	4.0

TABLE IV

Nationality	Investigator	No. Cases	Range of Percentage Findings O	A	B	AB
Stuttering group	Anderson, Whealdon	50	46.0	44.0	4.0	6.0
Swedish	Folgegren, Lindeberger, Hesser	1,533	33.5–36.9	46.9–51.0	9.1–10.0	5.5–7.2
German	Schiff, Wiechmann and Paal; Wagner and Kruse; Kruse, Leweringhaus, Fischer, Gundel, Arnold, Mayser, Corvin	about 50,000	29.5–43.0	39.7–45.9	9.6–18.1	2.5–8.5

Our finding, tested by this formula, tends to indicate that blood group distribution among these stutterers and the general population approaches parity.

[7]Garrett, Henry E. Statistics in Psychology and Education, pp. 213, 226–227. Longmans, Green Co., N. Y. 1937. This formula is also a method for determining the agreement of percentages, and our findings in this group of stutterers are in substantial agreement with those for the general population in view of the fact that there is no significant difference. Garrett's formula, applied to blood group B where the greatest deviation obtains, gives us a D/σ_{Dp} of 2.74, and Garrett states: "It is customary to take a D/σ_{Dp} of 3.0 as indicative of a significant difference (virtual certainty) since there is only about 1 chance in 1000 that a difference of +3.0 will arise when the true difference is zero."

Differences in groups A and AB may be partially explained when we consider the Scandinavian and Germanic backgrounds of over sixty per cent of our subjects. Blood group studies carried out in Sweden and in Germany indicate a higher percentage of A and AB types than have been found in the general American population. Perhaps it is this influence that tends to distort our findings from those found by other American investigators. It does not explain our low percentage of B types.[8]

Other studies have also failed to prove blood group linkage with specific diseases or tendencies toward them. Although slight deviations similar to those found in this study have been reported, no significant differences have been found in investigations concerning: schizophrenia and paranoia,[9] psychopathic disorders,[10–11] blood diseases and neoplasms,[12] poliomyelitis,[13–14], scarlet fever,[15–16–17–18] mongoloidism,[19] allergy,[20] cancer,[21] infectious diseases including scarlet fever, chickenpox, measles, whooping cough, diphtheria.[22] In the few instances where investigators have reported significant differences, later studies involving more subjects have disproved their findings.

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[8]Wiener, Alexander S., *op. cit.*, pp. 155–58.

[9]Prokop, J.; Skalickova, O.; Cupik, J. Contribution aux études Biotypologiques dans la Schizophrénie, les Groupes Sanguins, la Constitution et la Race. L'Encéphale: 2: 104–108. July-Aug., 1938.

[10]Herman, Morris and Derby, Irving M. The Blood Groups and M-N Type in Mental Diseases. Journal of Immunology: 33: 87–99. Aug., 1937.

[11]Thomas, John C. and Hewitt, E. J. C. Blood Groups in Health and in Mental Disease. Journal of Mental Science: 85: 667–688. July, 1939.

[12]Macleod, Kenneth I. E. The Relation of the Blood Group of the Individual to Blood Diseases and Neoplasms. British Medical Journal: 2: 745–747. Oct. 16, 1937.

[13]Erb, I. H.; Doyle, H. S.; Heal, F. C. Blood Groups in Poliomyelitis. Canadian Public Health Journal: 29: 441–442. Sept., 1938.

[14]Madsen, Th.; Engle, E. T.; Jensen, Klaus; Freuchen, Ib. Blood Grouping and Poliomyelitis Report based on 1118 cases in the 1934 epidemic in Denmark. Journal of Immunology: 30: 213–219. March, 1936.

[15]Bernecker, Liselotte. Die Blutgruppenverteilung bei Scharlach-Kranken. Monatschrift fur Kinderheilkunde: 66: 391–396. June, 1936.

[16]Vosschulte, A. and Ziegler, K. Zur Frage Blutgruppe und Scharlach. Deutsche Medizinische Wochenschrift: 61: 262–263. Feb. 15, 1935.

[17]Nowak, Herbert. Uber Blutgruppen und die Empfanglichkeit fur Diphtherie und Scharlach. Zeitschrift fur Rassen-Physiologie: 6: 154–156. Munchen, 1933.

[18]Brody, Henry; Smith, Lawrence W.; Wolff, William I. Blood Grouping in the infectious Diseases. Journal of Laboratory and Clinical Medicine: 21: 705–710. April, 1936.

[19]Caminopetros, J. Récherches sur l'anémie érythroblastique infantile des peuples de la Méditérranée Orientale. Annales de Médicine: 43: 104–125. February, 1938.

[20]Dyrenforth, Lucien Y. Blood Groups and Allergy: A Statistical Review. Southern Medical Journal: 29: 617–618. June, 1936.

[21]Kukowa, A. Die Blutgruppenverteilung bei Krebzkranken. Zeitschrift fur Krubsforschung: 42: 510–511. 1935.

[22]Brody, Henry et al., *op. cit.*

We can conclude only from our study of fifty stutterers matched against 38,000 individuals from the general population that there is an approximate parity of distribution among the stuttering and non-stuttering groups. This does not mean necessarily that there are no blood differences. It means only that there are no differences in these protein factors which govern red cell agglutination. There may be other differences of protein structure or of blood protein reaction with other blood substances or in the form of the protein present. Perhaps the different metabolism in stutterers suggested by Kopp[23–24] has something to do with our problem. We have yet to hit upon a way of showing whether or not these suggested differences do exist. Until we have shown that they either do or do not, it is not for us to formulate any conclusions other than the one already stated. We must also remember that not only in our study but in all those undertaken to show linkage between blood groups and a disease or tendency, we have no way of knowing whether or not the subject will contract the disease later—if he will break over his margins of safety into stuttering, for example. Moreover, in most of the investigations either no family histories were secured or the investigators ran into the difficulty of obtaining adequate information concerning the presence of diatheses in the families.

"One does not get nearer to the ideal state any the more quickly by trying to force the shortest cut to the goal of strictest unity, by proclaiming meager beginnings as the final principles, and by owing to the facts what is saved on the theories."[25] We have made a meager beginning. Lest we be owing too much to the facts we hasten to suggest that before final principles can be formulated, a study of blood crystallization in stutterers might be undertaken;[26] intelligence tests might be applied to a large group of stutterers; a study of M and N factors in the blood of stutterers might be made; a study of histories and mating combinations of blood groups in stuttering families might be attempted. These studies would make the beginnings less meager and might help to erect a signpost or two along the long and tortuous road leading toward a solution of the elusive etiology of stuttering.

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[23]Kopp, George E. Metabolic Studies of Stutterers. Speech Monographs: 1: No. 1. September, 1934.

[24]Kopp, George E. The Metabolism of the Stutterer as evidenced by biochemical studies of blood, alveolar air and urine. p. 162. Doctoral dissertation, University of Wisconsin, 1933.

[25]Kohler, W. The Mentality of Apes. Harcourt Brace and Co., N. Y., 1925.

[26]Rascher, S. and Trumpp, J. Attempt at Crystallographic Diagnosis of Carcinoma. Reported in Journal of the American Medical Association. 113: 3: 273. *From* Munchener medizinische Wochenschrift: 86: 544. April 7, 1939.

A STUDY OF THE EFFECT OF REMEDIAL TREATMENT ON THE FREQUENCY OF STUTTERING IN ORAL READING

EUGENE F. HAHN
Wayne University

It was the problem of this study to investigate the effects of certain remedial measures upon the speech of stutterers. Since little research has been conducted on this problem the validity of certain stuttering correction methods has not been established. The comparison of stuttering frequencies before and after remedial treatment requires comparable test situations, an established time limit for the training, and a record of the methods in use.

An earlier study[1] discussed the relationship between the social complexity of the oral reading situation and the severity of stuttering. In this investigation a group of stutterers was tested in four oral reading situations. The reading material used in testing consisted of four matched selections. In situation one the subject read the material aloud, alone in a small room. In situation two he read the second selection aloud after being told that the tester would stand behind an obstruction in the room, unable to see or to be seen by the stutterer, but able to hear the reading. In the third situation the subject read directly to the tester. In the fourth, he read in the classroom before a small group of stutterers and non-stutterers. During the first three situations a concealed observer recorded the occurrence of spasms. This observer was also present in the audience in the fourth situation.

The results of the study indicated:

(1) Stutterers experience significant stuttering difficulty while reading orally alone.

(2) Stuttering increases significantly when stutterers read aloud to an unseen listener, known to be concealed in the room, than when they read alone.

(3) Stuttering frequency varies little from a situation in which stutterers read directly to a listener and a situation in which stutterers read to an unseen listener.

(4) Stutterers experience a significantly greater number of spasms in reading before a group audience than they experience either in reading alone or in reading before an unseen or a direct listener.

(5) The variation in the number of spasms was significantly more affected by individual differences than by differences in the social complexity of the reading situations.[2]

[1]See *Journal of Speech Disorders*, V. 5, No. 1, March, 1940.

[2]The variations in the number of spasms due to individual differences was eight times larger than that due to differences in the complexity of the social situation.

After three months of remedial treatment, twenty-two of the adult stutterers who had read in the initial test situations were retested to ascertain any differences in stuttering frequencies. The retests duplicated the procedures of the initial tests. Thus, the occurrences of stuttering spasms of these stutterers in the first and in the retest situations could be compared to determine the effect of the remedial speech treatment.

Specifically, certain questions arose to which it was hoped this study would furnish answers.

1. Will the frequency of stuttering in the retests be less than the frequency in the initial tests?
2. Will the comparison of the results of the retests and the initial tests be statistically significant?
3. Will there be visible cues which the concealed observer can record which will give indications as to the stutterers' responses in the retest situations and which can be compared to these same stutterers' responses in the initial situations?
4. Will it appear feasible to advocate certain remedial measures as valuable in the treatment of stuttering?

Remedial Procedures

Organization of the class for stutterers. The subjects upon whom retests were made in this study registered at the University of Southern California for a course entitled "Corrective Re-Training of Stutterers." This class or clinic taught by the investigator was scheduled two evenings a week on a quarterly basis. Registration was limited to approximately fifteen persons a quarter. The basic philosophy in the management of clinical cases approximates that advocated by Mabel F. Gifford, chief of the Bureau for the Correction of Speech Defects and Disorders, State Department of Education, California.(3)

Fundamental concepts upon which clinical procedures were based. The major concept upon which the clinical procedures were based was that it is possible for the stutterer to speak without stuttering. This unqualified statement does not necessarily presuppose absolute cure. It means that in contact with society the individual is capable of speech which will not be so manifestly different from the normal as to be conspicuous or unpleasant. It is granted that the adult who has stuttered from childhood may return to his stuttering under conditions involving physical and emotional strain. Even for this individual the stuttering following such crises need not continue, for he may be brought back to successful speech responses by insight into the cause of his relapse and by the continuation of the re-education program. This present study does not state that a cure or "normal speech" was attained in any given case. The ability to speak without stuttering may be brought about by such measures as: bodily relaxation, physical control, emotional control, disruption of old habit patterns of speaking and substitution of a new speech

pattern, positive suggestion, and change of attitudes. These measures were emphasized in retraining the stutterers in the speech clinic.

The importance of relaxation was stressed for individual and group work. By means of it, one is able to reduce physical and emotional tensions and consequently can exert a higher level of conscious control over his body. Relaxation is the basic step without which the necessary measures cannot be put into practice. The term, "physical control," means conscious control of the body's activities. The realization of an intellectual conscious control over such emotional reactions as excessive fear, timidity, and embarrassment is a necessary correllary of physical control.

The reason for disrupting the old habit patterns of speaking is that the old patterns are faulty and are associated with speech failure. The use of the new speech pattern permits the stutterer to correct these faults and to associate success with his speech responses. The principle of positive suggestion is advocated. The stutterer, during the greater part of his life, has been subject to negative autosuggestion because his attention has been directed to his repeated speech failures, and he expects the re-occurrence of such failures. He is now directed to use positive autosuggestion. Johnson (1) recommends the use of legitimate suggestion based on demonstrable facts as to the stutterer's speaking ability and his increasing mastery over speech. In the clinic, the stutterer is asked to recall his successful speech responses in order to change his attitude from one of expectation of failure to one of anticipation of success. Another method of suggestion involves the constructive use of imagination. A stutterer is directed to visualize in detail one of his more difficult speaking situations and then to see himself speaking successfully in that situation. The changing of attitudes involves a mental hygiene program. Certain feeling states become habitual with the stutterer and influence his behavior. Those attitudes which prevent proper social adjustment need to be re-evaluated and supplanted by feeling states which allow the individual to approach the social situation with the maximum of emotional and physical control.

Of these various measures presented, the discussion of the speech pattern needs amplification. The pattern can be considered from two points of view: the stutterer's and the observer's. The stutterer should conceive of the pattern as involving an anticipation of success. He should achieve a feeling of complete bodily relaxation and of emotional stability. In speaking, he should have a feeling of pouring out the tone on the outgoing breath and he should emphasize the length of vowel sounds. He should speak in short phrases, pausing between phrases to replenish his breath and to re-establish the feeling of physical and emotional relaxation. The broken speech rhythm is then supplanted by an easy, rhythmical, controlled speech pattern. To the observer the stutterer's speech is characterized by a soft, breathy-tone quality, prolonged vowel sounds, short phrases, frequent pauses, and natural inflections. The rhythm is definite and rather slow. The bodily tensions and strains so prevalent among those who stutter are replaced by increased ease and calm.

The stutterer must realize that there is no effort involved in pouring out the breath. The breathing is natural but more regular than that required in the usual speech responses.

The question frequently arises in the clinic as to the advisability of all stutterers using the breathy tone in speaking. The use of breathiness alone does not eliminate the stuttering spasms, but the breathiness should be viewed as one element necessary in building up the totality of the pattern. More important than the amount of breath used in speaking is the psychological and physiological feeling that speech is being produced on the rhythmic, outgoing breath. Frequently, it is advisable to ask all stutterers to overemphasize the breathiness in the beginning stages of the clinical work, so that they will understand the action of the speech mechanism and perceive the feeling of the pouring out of the breath. Also, the average stutterer will tend to compromise on the amount of breathiness he uses in speech in outside situations. The exaggeration in the clinic allows for this anticipated compromise. One advantage in advocating the breathy tone is that the stutterer is given something specific and tangible to do, an action well within his field of understanding and obvious in execution to the visual, auditory, and kinaesthetic senses. One factor which helps to mitigate the unnaturalness in the use of the pattern is that the inflections of normal speech are required.

Every stutterer is not asked to emphasize all elements of the pattern equally. Each stresses those factors which meet his particular speech needs. For example, one stutterer may have his attention directed toward the use of passive mouth action in speaking; another may concentrate on the necessity of speaking in short phrases; and yet another may emphasize the length of vowels. Nor is it necessary that the stutterer's attention remain on one element in the pattern over a long period of time. His emphasis may change from week to week in an effort to establish the pattern as a whole.

In discussing the measures upon which remedial work in the clinic was based, one cannot overlook the fact that there may be operating phenomena not present among the measures treated. Also, it has not been established by research that one or more of these measures is more important than the others in affecting speech improvement.

Another basic concept in the treatment of stutterers is that it is possible for a stutterer to proceed from success in a simple speech situation to success in a complex social situation. Only by means of increasing the difficulty in the social situations can the stutterer finally be successful in satisfactory social adjustment to situations outside the clinic.

Detailed clinical procedures. The following speech correction techniques were used in the class during this three-month period of retraining:

1. *The attainment of bodily relaxation.* In the clinic lectures were given on the value of relaxation in speech situations. During the first meetings of the class, the instructor sat in front of the group and

began each period by asking class members to close their eyes, place both feet on the floor, drop the hands in the lap, and turn the attention first to the relaxation of the grosser muscles, and then to the finer muscles. The room was darkened. The time usually allotted for relaxation was fifteen minutes. The students were asked to read Jacobson's book on progressive relaxation (4). After four class meetings, the students carried on their own relaxation period before each meeting. No conversation was allowed. Any visitors were asked to co-operate with the group during this period.

2. *The establishment of sound on the outgoing breath.* Information and demonstration were given on the method of producing speech. The instructor called attention to the psychological feeling of "pouring out" the tone on the breath. He demonstrated the procedure by producing each long vowel on the outgoing breath. The class repeated the exercise in unison. The instructor demonstrated all the vowels on one breath and asked for group response. Individual response on vowel sounds followed. In order to maintain the feeling of relaxation and to give closer attention to the significance of the outgoing breath, the class members kept their eyes closed and the room darkened. Volunteers were then asked to stand before the group and to produce the vowel sounds. During this time, the instructor made notes concerning those who volunteered and those who responded most successfully in producing the correct breathy tone. Individual responses were repeated with the full lights turned on in the classroom. Usually these procedures comprised the first few meetings of the stuttering class.

3. *The use of silent recall.* In using recall, the stutterers were asked to produce vowel sounds with the eyes closed and then to recall the sensation of that production of the vowel on the "out-pouring" breath. This established the kinaesthetic feeling of ease of vocal production. Silent recall was also used for autosuggestion in building up confidence that a sound could be repeated successfully.

4. *The establishment of words and phrases on the out-going breath.* Single words and short phrases were produced on the outgoing breath in the same manner in which vowels were produced. Individuals, in answer to questions from the instructor, replied in single words or short phrases, using the breathy tone. The instructor asked questions pertaining to name, address, phone number, date and place of birth, date and name of day, directions for reaching the school, names of persons present, description of wearing apparel of class members, and so forth. After the first few class meetings the instructor always called for the group response of vowels on one breath and then individual responses on names and addresses. Those students who were not able to make individual responses successfully were given additional practice in another room by an assistant.

5. *The use of passive and loose mouth action.* The value and significance of speaking with a very passive, relaxed mouth action was explained to the stutterers. The loose mouth action was demonstrated. The instructor spoke with the jaw hanging loosely, with no oral action in speaking except that of the tongue. Even with such

exaggerated relaxation, speech was understandable. The passive mouth action was explained as involving normal articulation with a minimum of effort. Those individuals whose forward pattern of speech was inhibited by tonic blocks were directed to use the loose mouth action. All stutterers were asked to use passive mouth action because it entailed a high conscious level of attention on the ease of speech production. The loose and passive mouth action were used to eliminate prevalent difficulty with consonant blocks.

6. *The establishment of pauses in speech.* To maintain a high cortical level of control over his speech responses the stutterer was directed to master the use of pauses in reading and speaking. Poetry and prose selections were given and a stop watch was used by the instructor to ascertain the length of pauses. The individual was reminded about the importance of complete bodily control during these pauses.

7. *The prolongation of vowel sounds in speaking.* In order to maintain a feeling of control over his speech mechanism the stutterer was directed to lengthen vowel sounds. The rate-control technique recommended by Bender and Kleinfeld (2) was used occasionally.

8. *The technique of merging speech situations from the simple to the complex.* In class work the responses varied from group response to individual response within the group to individual responses in front of the group. Occasionally, two or more persons participated in front of the group in one project, such as the staging of informal conversations. Sometimes the class was divided into two groups seated at different tables and each person was allowed to ask three questions of the stutterer at the head of the table. Again, a student turned to his left and asked another member a question. In another project one stutterer stood beside another, obtained a question from him, and repeated the question for the group in a louder voice. He also obtained the answer from another student and repeated that answer for the larger audience's benefit. As the class progressed, the situations became more difficult. Members were seated in the center of the room and were cross-examined by the instructor on a given subject. Often members were grouped two by two for conversation. At five-minute intervals they changed partners. During this time, the instructor walked around the room and listened to the responses of individual members to discover how well they were using the speech pattern. Varying situations were also evolved to meet individual needs. From these situations within one room the next step was the use of the multiple-room technique. Finally, after much practice the stutterers presented five-minute speeches before the class group and frequently before a strange audience. This project was assigned near the climax of the retraining period. It was not uncommon during the course to have visitors. These numbered from one to two at the beginning, and frequently reached a total of twenty-five at the end of the course. When visitors were present, it was the usual procedure for the stutterers to introduce themselves to the visitors and to introduce fellow class members.

9. *The use of the multiple-room technique.* Occasionally, stutterers

in small groups, segregated according to their stuttering difficulty. They were sent to different rooms and worked on specific projects to meet their individual needs. Occasionally, one-to-two-minute speeches were assigned and students progressed from one room to another, giving their speeches before the small groups. Sometimes a stutterer from one of the groups experiencing little speech difficulty was sent to one of the less advanced groups to give recommendations and practice. Often as many as six rooms were in use at one time. Radio equipment was used in the following manner: stutterers were sent into the recording room alone and in small groups to practice the use of this speech pattern; they were able to hear the broadcast as they spoke; again, they read, spoke, and carried on dialogue, broadcasting to the main classroom.

10. *The establishment of the use of the pattern in situations outside the clinic.* As a starting point in carrying over the speech pattern into outside situations each stutterer was directed to find a speech situation so simple that it was not associated with stuttering difficulty. Then, just as in the clinic, these situations were emerged from simple to complex. For example, if a stutterer could talk to a young child without stuttering, that was the first situation in which he put the pattern into use. He might stutter in other situations during the day, but he always returned to this situation to re-establish the conviction that it was possible for him to speak without stuttering. In the weeks that followed each stutterer handed in reports showing his progress from the simple situation to one which was more complex for him. Near the end of the course each student handed in complete situation reports. The stutterer was asked to write up two situations a day in which he had deliberately created the speech situation, such as, for example, making a purchase from a store. In writing this up he followed an outline similar to this:

1. Name and place of person with whom conversation was held.
2. Statement of planned conversation.
3. Stutterer's reaction to situation before, during, and after conversation.
4. Evaluation of speech success or failure. Each stutterer was asked to grade himself three times a day. All stutterers who entered the clinic were considered as having a grade of C in speech, that is, they were doing their average amount of stuttering. If during a given day they felt that they had improved, they graded their speech B; if to their knowledge they did not stutter, they gave themselves an A; if their stuttering appeared worse, the grade was D. If there was any great fluctuation in marking, the cause was analyzed in consultations with the instructor.

11. *The establishment of personal contact between student and instructor.* Each student was requested to write an autobiography to hand in to the instructor. This was used as the basis for private consultation. Stutterers were also given Humm-Wadsworth (5) Temperament Scale tests and the results of these provided data for personal discussion. The grading and situation reports were also considered by instructor and individual in interviews.

It is recognized that the personality of the clinical director is an important factor in managing cases. Although this has not been subject to experimentation, it is commonly said that this personality factor is such a strong motivating force that remedial success depends upon it to a considerable extent. For the purpose of this investigation, one can only assume that the director motivated the stutterers to apply themselves as much as possible.

12. *The presentation of short lectures on mental hygiene.* The instructor presented short lectures on different aspects of mental hygiene from time to time during the retraining period.

TABLE I
COMPARISON OF PERCENTAGE* OF WORDS UPON WHICH STUTTERING OCCURRED IN INITIAL AND IN RETEST READING SITUATIONS FOLLOWING REMEDIAL WORK

Case Number	Initial Situations				Retest Situations			
	1	2	3	4	1	2	3	4
1 (f)	2.9	10.2	9.1	10.7	4.4	6.5	8.7	12.0
2	10.2	13.6	15.8	11.8	0.0	1.5	2.0	2.2
3	6.5	15.1	12.4	12.2	.2	3.8	1.8	3.6
4	3.6	10.1	10.9	27.8	0.0	0.0	.2	1.1†
9	2.5	3.8	2.7	2.7	1.1	1.1	.7	.5†
11	4.5	6.2	6.2	5.3	1.1	.9	.4	.2
12	.2	4.4	25.6	40.9	.2	.2	0.0	0.0
13	6.7	10.1	15.3	18.7	2.2	2.5	3.5	4.5†
15	.5	.4	.2	4.0	0.0	0.0	0.0	0.0
16	14.4	12.7	9.3	11.8	.4	.5	.7	1.6
17	1.5	5.5	9.3	14.5	0.0	.2	0.0	0.0
19	.2	18.4	2.9	4.0	0.0	0.0	0.0	0.0
20	10.4	22.7	17.5	27.6	.2	0.0	0.0	2.0
21	.5	.2	1.1	1.5	0.0	0.0	0.0	.7†
22	11.6	15.6	18.4	20.4	0.0	.9	.9	7.6
29 (f)	3.1	4.4	4.9	9.3	.2	.4	.5	.7
31	7.1	18.9	18.4	18.5	0.0	0.0	0.0	0.0
44 (f)	0.0	6.9	10.2	15.6†	0.0	1.1	3.1	6.9†
45 (f)	31.1	56.4	51.3	63.1†	1.1	8.0	15.5	29.6†
46	4.7	3.5	4.0	4.4†	1.1	1.6	1.1	.4
51	6.7	11.5	5.7	8.0†	0.0	.4	.2	1.8
52	2.9	5.1	10.4	14.2	1.1	.4	.4	0.0
Mean	6.0	11.6	11.9	15.8	.6	1.4	1.8	3.4
Median	4.1	10.0	6.7	12.0	.2	.5	.5	.9
S. D.	6.7	11.5	10.7	13.9	1.0	2.1	3.5	6.5

*Percentage is figured on basis of 550 words in reading selection.
†This percentage is estimated.

Results and Discussion

The results of the retesting in the four situations are shown in Table I. The percentage of words upon which stuttering occurred in each 550-word reading selection is presented both for the initial tests and for the retests. A comparison of the results of the initial and

retest situations shows that for these twenty-two stutterers the average number of stuttering spasms decreased from 33.0 to 3.3 in the first situation during which the stutterer read orally alone. The percentage of stuttering derceased from 6.0 to .6. In the second situation, reading to an unseen listener, the average number of spasms decreased from 63.8 to 7.5; the percentage of stuttering, from 11.6 to 1.4. In the third situation, reading directly to a single listener, the number decreased from 65.6 to 9.9; the percentage, from 11.9 to 1.8. In reading before the group audience, the stutterers' difficulty decreased from 86.8 to 18.9 spasms, from 15.8 to 3.4 per cent. In the first situation the number of spasms ranged from 0 to 171 in the initial tests, and from 0 to 24 in the retests. In the second situation the ranges were from 1 to 310, and from 0 to 44. In the third situation the ranges were from 1 to 282, and from 0 to 85. In reading before the group the stutterers had from 8 to an estimated 347 spasms in the initial tests, and from 0 to an estimated 163 in the retests.

In comparing the differences between the means of the stuttering frequencies in the four situations in terms of standard error of these differences, it is found that for Test Situation I, the ratio of the difference to the standard error is 3.74. The same ratios for Situations 2, 3, and 4 are: 4.56, 5.54, 5.46, respectively. A ratio of 3.0 is considered statistically significant. Since these ratios far exceed this figure, there is little probability that the decrease in spasms was due to chance fluctuation.

The correlation coefficients between stuttering frequency in the nitial test situations and in the retest situations are:

r_{IR} for Situation 1: .05
r_{IR} for Situation 2: .62
r_{IR} for Situation 3: .70
r_{IR} for Situation 4: .68

In comparing the results of the initial tests and those of the retests, again it is shown that the situations become progressively associated with increased stuttering; and, further, that Situations 2 and 3 are fairly comparable in amount of difficulty. The fourth situation more than doubles the first in the number of spasms.

In considering individual responses the concealed observer noted that most of the cases attempted to put the advocated speech pattern and the suggestions for speech improvement into practice in the reading situations. However, the success of individuals varied. Several cases had not yet mastered certain phases of the pattern. Case 1, for example, spoke in long phrases, with inadequate pauses, and had not yet learned to control the emotional excitability after responses. Case 13 did not attempt to use any of the suggestions given in the remedial treatment. Cases 22 and 44 stated that they were emotionally upset by the group audience. Case 45, the most pronounced stutterer, obviously needed a longer period of training.

It is hoped that in time, methods of testing speech more similar

to that used in real life situations and in testing improvement in such speech will be formulated

Summary

Twenty-two adult stutterers who had read the initial test selections were retested after three months of remedial work. The situations for the four tests were the same as those used in the initial tests: reading alone, reading before an unseen listener known to be present, reading directly to a single listener, and reading before a small group of stutterers and non-stutterers. A concealed observer again checked the number and location of stuttering spasms for each reading. The selections read were the same as those used in the initial tests.

In comparing the results of the initial test situations and those of the retest situations, one can only conclude that

1. Stuttering frequency can be significantly reduced by proper remedial treatment.

2. The remedial procedures thought to be responsible for this reduction are: bodily relaxation, physical control, emotional control, disruption of old habit patterns in speaking and the substitution of a new speech pattern, positive suggestion, and change of attitudes.

3. The use of the speech pattern and the objectifying of suggestions for emotional and bodily control given in the remedial measures are visible to the observer. According to the observer's notes, the use of such speech techniques is related to the decrease in the frequency of stuttering.

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SECTION ON

Elementary, Popular and Semi-Popular Articles

The policy which has built the present reputation of the JOURNAL OF SPEECH DISORDERS has been to favor sound scholarly articles adequately supported by objective evidence. Since it was initiated the membership of the association has grown by leaps and bounds. True, membership (though not fellowship) was opened to many with but meagre training in the hopes they would be stimulated to better preparation, yet it did grow phenomonally.

Shortly a strong movement was initiated in the name of democracy, however, urging more articles from and for this group, which should of necessity be more popular in nature. Your editor opposed any lowering of scholarly and scientific standards, and every effort was made to foster the elementary magazine, "Speech," to care for that demand. Likewise the association for elementary teachers which seemed to get off to such a good start at St. Louis a half a decade ago.

The facts seem to be though, that most such individuals prefer the prestige which even mere membership in the American Speech Correction Association gave them. For that elementary movement did not prosper in spite of the whole-hearted support we rendered. Meanwhile our membership and subscriptions continued to grow. Yet the movement to lower the standards for admission to fellowship and also those of articles published in the JOURNAL OF SPEECH DISORDERS likewise grew more and more vocal. Your editor and many others seriously question the wisdom of such a move. Those who think it represents a more democratic procedure to cater to the large groups it is true of those of those with but meagre preparation, to the unscientific and unscholarly, to the popularly minded, should be convinced by now of the unfortunate effect the movement has had.

Standards need to be raised in the profession, not lowered. If the American Medical Association had not followed the first policy, what would have been the result? It is well to remember that there are an appalling number of quacks treating stutterers alone. We cannot afford to foster even a semblance of a movement in that direction.

Then, too, a clear distinction should be drawn among those who belong to the Association, as between two groups:

1. Those qualified as SPEECH PATHOLOGISTS to treat serious speech disorders, especially where pathological impairments are involved. Obviously these specialists should be required to have a thorough preparation in pathology (as far as neuro-pathology), anatomy (to human dissection and neuro-anatomy), abnormal and clinical psychology, to say nothing of allied fields.

2. Those qualified as MINOR SPEECH CORRECTIONISTS. LOGOPAEDISTS and SPEECH THERAPISTS, they are called abroad. These need have taken no such severe courses to qualify as correctors of baby-talk, foreign accent, phonetic substitutions, and any bad speech traceable to wrong habits or poor training.

The rank of "fellow" was intended for the first. And that of "member" for the latter. Just as the title "doctor" is applied to the M.D. and "nurse" to the one with an equally honorable but quite different training.

Then there is your JOURNAL OF SPEECH DISORDERS. What of it? The doctors are numerous enough and can afford to publish any number of not only technical, sound scientific, and scholarly professional journals (all of which would be fearfully dull to those lacking the training to read them);

but also of *Hygeia*, a semi-popular publication designed purposely to be interesting. Or there are the scholarly journals, *Modern Language Notes*, and *Language*, to contrast with the *English Journal*, or one of the N. E. A. publications for elementary teachers.

The best we have been able to hope was that those who did not like our JOURNAL OF SPEECH DISORDERS and our Association policy would turn their support to the more popular *Speech*. We can get ample support and still maintain high scholarly and scientific standards. And too, it is to be hoped, gradually eliminate quackery in the field of Speech Correction.

This issue adds, however, another section as an experiment. Tentatively it is given the title "Elementary, Popular and Semi-popular Articles." Such would be those not accompanied by any objective proof a fellow worker could check for validity. Insertion of the articles listed is hardly fair to the authors. Neither is it to imply they were passed upon by the editor indicated for this section (indeed. by the way, without his permission). For he has seen only one of them. But for the forthcoming December issue he will provide the type of articles he feels he would like to see fostered and of the kind he called for from the floor of the last convention. Meanwhile the Journal editor would like to have an expression of opinion from members of the Association.

(Signed) G. OSCAR RUSSELL, *Editor*.

All future articles for this section will be sent directly to Professor Max Steer, Purdue University, Lafayette, Ind., who alone will assume responsibility for their publication.

The JOURNAL will furnish no reprints to authors of articles in this section.

STUTTERING: A TENTATIVE OUTLINE OF AN HYPOTHESIS AND THERAPY[1]

CLYDE W. DOW, M. S.
Massachusetts State College

During stuttering, says Dr. Solomon[2], we have "speech characterized by transient interruptions of the rhythm of speech, resulting in speech block, whether incomplete as in hesitation and repetition, or complete as in temporary inability to produce any sound or their combination."

It is not extremely difficult to obtain a fair amount of agreement on the above definition of the *act* of stuttering, but when you ask a group of speech correctionists the cause of stuttering and the method of treating it, agreement stops. We have a multitude of theories and therapies, sworn at by some, and sworn by in the case of others. Many of the theories seem reasonable, and many of the therapies—even those based on theories that seem unreasonable—appear to bring about improvement for the stutterer. Dr. Johnson[3] has gone so

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[1]This paper is part of a report for a Seminar in Speech Pathology with Dr. Robert West at the University of Wisconsin during the 1940 summer session. Dr. West is not to be held responsible, however, for any statement made in the paper.

[2]Solomon, M., "Stuttering as An Emotional and Personality Disorder," *Journal of Speech Disorders*, Dec., 1939, p. 347.

[3]Wendell Johnson, "The Treatment of Stuttering," *Journal of Speech Disorders*, June, 1939, p. 170.

far as to say: "The longer I work with stuttering the more tolerant I become of any one who has any therapeutic ideas at all concerning it. . . . . There is no such thing as *the* method for treating stuttering, and the fact that so many different methods are more or less successful is of far more than incidental interest."

A reasonable conclusion appears to require that we maintain, at present, that there is no single cause, no single theory, and no single therapy; there are causes, theories, and therapies. In line with these beliefs, the writer here outlines another approach to the understanding and treatment of stuttering.

Anyone who has worked with stuttering has frequently been dissatisfied with the theory, the progress of the subject, the results; and sometimes all three. Dr. George A. Kopp[4] makes a clear statement of this attitude that many of us have experienced.

> ". . . I have questioned every theory of stuttering that has ever been advanced. I have done this because I was convinced that no one method of treating stuttering is sufficient to correct the speech of all stutterers. Nor is any one method adequate to correct the speech of the majority of stutterers. In ten years of working with stutterers I have never been able to completely cure a single adult stutterer, using one or several approaches. It is true that I have helped to improve the speech of a number of stutterers, both young and old. But I would hesitate to assume much credit for what happened to any of them. Dr. West had an apt way of distributing the honors, if any, for what we did with stutterers at Wisconsin. He would say, 'Oh sure, God, nature, the patient, Kopp, and myself have helped a few stutterers. . .' "

The same sort of dissatisfaction has led the present writer to evolve a plan that may be incorporated into programs for the treatment of stuttering. The hypothesis and therapy are far from perfect as I have discovered in attempting to compare them with other theories and treatments[5] [6]. But I believe the plan worthy of further trial since there are indications that it is psychologically sound, and considerable progress has been made by some subjects when such training was added to other therapies. The outline is tentative, and is subject to change as new facts present themselves, and as different techniques may be incorporated into the therapy to make it more effective. The method is not a proved one; it is a trial one.

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[4]Kopp, George A., "Treatment of Stuttering," *Journal of Speech Disorders*, Jupe, 1939, p. 166.

[5]Considerable similarity will be found between several points of Bluemel's "Inhibition Theory" as presented in *Stammering and Allied Disorders*, Macmillan, N. Y., 1935.

[6]Compare also, Kenyon, E. L., "Conscious Detailed Psycho-Muscular Control of Speech Production as an Effective (If Not Even Necessary) Basis for All Manner of Psychologic Treatment of Stuttering," *Symposium on Stuttering*, American Society for the Study of Disorders of Speech, Ed. by Robert West, 1930, pp. 176–187.

Certain obvious points about speech should be recalled before a statement of the hypothesis is made: The speech of the average person is acquired quite "unconsciously." Through trial and error, that begins in early childhood, the human being gradually builds up audio-kinesthetic sensations that result in the refinement of speech. When the average person talks, he does not think of making sounds; he merely uses a convenient, socially acquired, method to convey his meaning or wishes or to achieve his purpose. The stutterer "learns" his speech in the same way. He does not become aware of the various positions of the articulatory organs nor of the complicated mechanism involved in the production of speech sounds. When he stutters, he is probably quite unaware of just what he is trying to do. He knows that he is trying to talk, but that is about all. And the observer sees the phenomenon that we call blocking. At times, when the stutterer attempts to persevere or to force through the utterance of his thought, a more severe block may take place; and at times, we get what may be called secondary or associated symptoms such as facial grimaces, tics, and other physical manifestations. These various physical manifestations may, by repetition, become habitual accompaniments of the act of speaking.

Why this should happen to some people and not to others I do not know, but I venture the reason that some nervous systems are apparently more strongly constructed than others and learning arrangements (of the neurones) are more stable than in others. Bluemel[7] has suggested that the conditioned reflex of speech is more strongly established in the female than in the male. It may be reasonably true that the conditioned reflex of speech is more firmly established in some people than in others regardless of the sex.

This hypothesis assumes that in normal speech there is a nerve current from the appropriate cortical region to the appropriate muscle groups that is carried out with ease and without interruption or impedance; i. e., the nerve current may flow easily through the appropriate neurones to bring about the muscular response.

The hypothesis assumes that in the stutterer's speech the efferent nerve impulse (from brain to muscles) is impeded, and prevented from carrying out its appropriate action. If the impulse is unable to complete its action there may be a synpatic spread that will innervate other neurones leading to other muscles or to other parts of the same muscle, and we have the outward physical manifestations of stuttering.

Where the impulse to carry out the speech act is impeded and what impeded it, it is impossible to say at the present time. There may be a blocking or stoppage in the muscles themselves; it may be in the efferent nerve, or it may be in the cortex. The writer does not know what happens in the central nervous system, but there are possibilities that any impedance can cause a spreading through synaptic connections and thus bring about complicated disturbances.

The influence of emotions must also be included in this picture.

[7]Bluemel, C. S., *Stammering and Allied Disorders.*

Just how the emotions affect the central nervous system we do not know, but there seems to be ample ground for assuming that emotions may have either a stimulating or an inhibiting effect. Under some emotions, people do not think as effectively as normally, nor do they execute muscle movements with the same skill. Apparently the emotions affect the neural impulse (probably at the synapse) and assist or retard the traveling of that impulse.

Theory Underlying Treatment

In speech learning a neural arrangement is made from the cortex to the appropriate muscles, and it is along this arrangement of neurones that the impulse moves to bring about the innervating of the muscles. Since the complex physical-electical impulse can flow easily it goes direct to the appropriate muscles. ("Thus, for a given afferent neuron there is a path of least resistance through the potential synapses."[8] In the stutterer the impulse is impeded and so moves in other directions. ("More vigorous stimulation may, however, enable the stimulus to spread to additional neurons and thus arouse a greater response"[9]) What we shall try to do therefore, is to establish an easy flowing neural impulse to the appropriate muscles, so that there will be no necessity for that impulse to find an outlet over other neural connections. We shall follow the recency-frequency theory: where energy has gone most often it will go most easily, and we shall try to get that energy (the neural impulse) to flow direct to the appropriate speech muscles. ("The whole process of the training of the neuron system seems to depend upon the fact that the more often a stimulus is routed through one pathway, the lower becomes the resistance of the synapses along this pathway and therefor the easier it becomes to arouse the particular reflex arc again."[10])

Therapy

The therapy, based upon the hypothesis given above is as follows: By a drill technique we shall try to set up an appropriate arrangement of neurones (the same as happens in any learning process) and we shall try to make that new-learned response a habit eventually; i. e., try to make it take place "unconsciously" in due time.

When a stutterer comes to a block, he is, as we have noted above, quite unaware of just what physical adjustments he is trying to make. He is against an impassable barrier, and no solution is apparent to him. Since he is frustrated and cannot adjust to the situation, emotions arise that prevent an intelligent approach to the problem. So the method of treatment should strive to show *how* the situation may be met and the emotion prevented.

(a) The first step in the program is to explain the hypothesis as given above. Be sure that the subject understands it and try to get him to believe it. Suggestion probably has some important influences

[8]Eisenson, J., *The Psychology of Speech*, F. S. Crofts, N. Y., 1938, p. 40.
[9]Eisenson, J., *The Psychology of Speech*, p. 40.
[10]Eisenson, J., *The Psychology of Speech*, F. S. Crofts, N. Y., 1938, p. 40.

here, but it is impossible to state *how* important. (b) Next, the subject learns simplified definitions for the positions of the articulatory organs when making various sounds. This is a learning-while-doing process. The subject and the instructor work out simple definitions by trial. The procedure here is much more simple than that used by Dr. Kenyon: In most cases, two or three fifty-minute periods are sufficient to acquire the necessary definitions for all sounds. (c) There are drill periods during which the subject and the instructor review the definitions and the muscle positions and movements involved in the various sounds. The instructor may ask how the sound *d* is made, and the subject gives the definition and then performs the muscular adjustment. (d) As soon as possible poetry and prose are read aloud, and then short prepared talks are given. In the early part of the training, when the subject encounters a block, the instructor usually interrupts and taking the sound on which the block occurred says, "You're having trouble with *G*. How is *G* made?" The subject gives the short definition he has learned and then produces the sound. He then says the word and continues his reading or conversation. (e) Later, and at all times outside of class, the subject is advised to use a modification of the procedure in (d) above. Whenever a block occurs he is told to stop immediately and "mentally" or sub-vocally give himself directions on how the sound is made; then to make the sound and continue his conversation (f) In conjunction with the procedures above the instructor also uses many techniques used by other speech correctionists. " . . . Therapies as they are practiced are seldom limited to a single approach such as the physiological, psychological, sociological, neurological, psychoanalytical, pedagogical, or any other point of view. This is as it should be, but why claim credit for one method when several are being used? The fact that in practice we recognize the merit of other methods is important to us today".[11]) These other techniques are of great value and the contribution they make to the total treatment is not meant to be disregarded, but I wish to emphasize the first five points included in this therapy. Some other techniques include the planning of a program of social activities, a program for adequate rest and recreation, suggestions for relaxation, and attempts to get the subject to speak slowly. Encouragement is given as often as reasonably possible. The subject is advised to live a reasonable normal life: to participate in class discussion; to go right ahead as if he had no speech difficulty, and if he does encounter it to use the techniques he has acquired in class.

A special mention should be made that stuttering may occur on any sound. The reason individual sounds are used for the approach to the problem is to give the stutterer an immediate attack upon his problem; to allow the neural impulse to follow the path we want it to take; and to establish a habit at the earliest possible opportunity so that any successes will tend to strengthen the neural bond.

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[11]Kopp, George A., "Treatment of Stuttering," *Journal of Speech Disorders*, June, 1939, p. 166.

The whole method outlined above may be working on entirely different grounds than that supposed by the writer. Perhaps the subject "gains confidence," "learns to make a satisfactory social adjustment," "establishes dominance," or gains "a feeling of inner calm," as other writers have maintained. Perhaps we may be able to check some of the theories some day. The therapy seems to the writer to be based on good habit techniques, and to be simple. Simplicity does not necessarily make a good plan, but it does make it easy for the stutterer to use. And, "regardless of the specific clinical techniques we may use, our aim is either to alter the so-called stuttering behavior by means of directed instructions, or to alter it indirectly by altering the stutterer's assumptions regarding it. . ."[12]

DIFFERENCES IN THE SYMPTOMATOLOGY OF STUTTERING IN THE U. S. AND IN EUROPE

DR. EMIL FROESCHELS
New York City

Stuttering is a nervousness, which as a rule rises in early childhood. Nervousness, in my opinion, is based on the readiness of the soul to fix pathological symptoms. This readiness seems to be a subconscious tendency to resist the difficulties or hardship of life. The nervousness called stuttering differs from other forms of nervousness in the fact that its symptoms are always subject to self observation. In other words, the patient can notice every false movement of the articulative muscles. This fact is the main reason why some therapists try to cure the disease with speech exercises alone. It is, however, my opinion that only a combined psychological and phonetic treatment is effective.

A brief description of the beginning and development of the impediment may help to explain my theory about the nature of stuttering. At the age of four, five or six years many children are repeating syllables during fluent speech. Observation has shown, that they do so, at least at the beginning of the impediment, if they don't find the next word, they want to pronounce. Incongruity between the speech temperament and the vocabulary or the grammatical skill is the basis of those iterations. Most children lose this incongruity after a couple of months and the iterations persist in 1 or 2 per cent only. These children are, as I believe, apt to fix abnormal symptoms. After a few weeks or months another symptom appears, consisting in an exaggerated force of articulation, which

[12]Johnson, Wendell, "The Treatment of Stuttering," *Journal of Speech Disorders*, June, 1939, p. 172.

is joined with iterations. We explain this symptom as an intention to overcome the abnormal movements, namely the iterations. The child is often assisted in this attempt by his environment, which misunderstands the real source of the syllable repetition and believes it to be a difficulty of pronunciation. Sooner or later accompanying movements, such as grimaces, clenching the fists, contractions in the legs or arms, and so on, will appear. Accompanying movements are also physiologically a sign of strain. The accompanying movements may be like primary stuttering movements either clonic (repeated contractions) or tonic (pressure). There is but one arbitrarily movable group of muscles in the body, which reduces only the tonic form of accompanying movements in stuttering, the muscles that move the eyeballs. Why does the clonic form, which is known as nystagmus, not appear as an accompanying movement in stuttering, but does appear in all other movable muscles? Because allother muscles can be voluntarily used in the form of permanent or repeated contractions. But the eye muscles can only be contracted permanently under the influence of the will. *That means, that only those movements are symptoms of stuttering, the performance of which depends upon the will.* This indubitable fact should be a memento for the understanding of the speech impediment mentioned. *If all symptoms are joined with the will, the whole disease must consequently be a subconscious action of the will.*

The pressure in the speaking muscles becomes by and by a symptom in itself, besides simple iterations, iterations joined with pressure and accompanying movements.

In Europe I had the opportunity to observe about 15,000 stutterers and I found that around the time of puberty the obvious symptoms were, as a rule replaced by less obvious; e. g., a stutterer of 15 years did not show, or showed only very seldom ugly grimaces, a wide opening of the mouth, protrusion of the tongue, and so on. He avoided looking into the eyes of his partner, showed a wax-like face and hid his clenched fist in his pocket. I called this stage "*Hidden Stuttering.*"

In the speech clinics in St. Louis and Ann Arbor I experienced that stutterers *in the United States don't get, or get much later, into this stage.*

If we look for an explanation of this fact, we can find it in the different opinion concerning the basis and the curability of stuttering among the patients here and in Europe. By inquiring I learned to know that many of them were convinced of an organic cause of their speech impediment, and therefore very skeptical with respect to the possibility of being cured. This opinion did not prevail in Europe.

Regarding the fact that the confidence in the curability of stuttering helps the therapist, I greatly appreciate the tendency of many American writers and therapists, to avoid bringing the theory of an organic basis into the compass of laymen. I appreciate this tendency as much more as my experience seems to give proof of the *psychogenesis* alone of stuttering.

THE SPEECH CLINIC AS A SOCIAL AGENCY IN NATIONAL EMERGENCIES

(A digest of a symposium-forum held on December 30 as a part of the program for the convention of the National Association of Teachers of Speech in session at Washington, D. C.)

Program sponsor: H. Koepp-Baker, Pennsylvania State College.

Presiding: I. S. Wile, New York City.

Members of the forum: Emil Froeschels, New York City; Alonzo J. Morley, Brigham Young University and Claude E. Kentner, Louisiana State University.

Discussion from the floor by: M. D. Steer, Purdue University and Charlotte Wells, University of Wisconsin.

The forum was called to order by the chairman who asked Mr. Froeschels to open the discussion. Mr. Froeschels spoke at length of his war time experiences as Director of Speech Clinics in the University and in the General Hospital in Vienna. He stressed the dual role of the speech clinician in the care of war-wounded and shell-shocked soldiers and civilians. He pointed out first that the speech clinician serves as a consultant to the neurologist in the diagnosis of the case and in determining the location of the lesion in the nervous system. The speech examination is often of great help to the neurologist, especially in aphasic cases, in locating the bullet or shell fragment in the brain. Mr. Froeschels stressed also the importance of the analysis of the speech specialist in differentiating between hysterical speech disturbances caused by shock and those speech defects arising from organic lesions. The second role of the speech clinician is his function as director of the remedial training. Upon him falls the responsibility for the speech re-education of the patient after the neurologist and surgeon have done their work. He illustrated his discussion with slides showing diagrams of various disturbances and pictures of some of his cases.

The discussion was continued by Mr. Morley who spoke of the need for co-operation between the speech clinic, the mental hygiene clinic, the vocational guidance clinic and other similar social agencies. He pointed out that the stresses and strains of war time conditions cause an increase in speech defects as well as in psychological disturbances and social and vocational maladjustments. The speech clinic needs to be equipped and ready to handle these conditions and particularly to co-operate with other agencies that are attempting to deal with the same problems.

Mr. Kantner then spoke briefly to the effect that war time emer-

gencies inevitably bring on financial restrictions and a curtailing of many peace time activities. This is particularly true in educational institutions, and speech and speech correction are still regarded by many as a luxury to be dispensed with in time of emergency. He concluded that many speech clinicians working in college and public school clinics are not in a position to make any direct and important contributions in war time emergencies, and that consequently their chief concern must be to maintain their work on as nearly a normal basis as possible and to so demonstrate its value to those in authority that it will not be curtailed in an attempt to meet financial problems arising out of war conditions.

At this point, the chairman summarized the discussion and remarked that this seemed to be a propitious time for a vigorous expansion of speech correction work. He called for discussion from the floor and Mr. Steer described briefly the efforts of a committee of the American Speech Correction Association to meet authorities directing the preparedness program and if possible to secure an official place for speech correction as a part of the activities in the various training camps in this country. Miss Wells discussed the role of the speech correctionist in the unifying of public sentiment and the promotion of Americanism through the elimination of foreign accent, thus bringing about a more homogeneous American speech. After brief concluding remarks by the chairman and the members of the panel, the forum was adjourned.

Claude E. Kantner.

March 3, 1941.

As an addendum to a recent article of mine in the "Journal" ("A Survey of Speech Defectives in the Indianapolis Primary Grades," September, 1940) the assumption was present that the situation revealed was as of the publication date.

Since the data was gathered for this survey (1937), the Indianapolis Public Schools have rightfully set about to correct this need. The present director of Speech Correction is Miss Esther Glaspey, a well-trained speech pathologist. Her progressive remedial speech program in the Indianapolis Public Schools has met with hearty approbation.

L. Gray Burdin.

SECTION ON

TECHNICS, UNIQUE CASE HISTORIES AND DISCUSSIONS

Edited by

MARTIN F. PALMER,

Director of the Institute of Logopedics

University of Wichita, Wichita, Kansas

(All contributions submitted for publication in this department should be sent directly to Dr. Palmer at the above address. That includes short disagreements with facts or conclusions in articles published by the Journal. Articles longer than two pages will be sent to the Editor of the Journal however.)

HISTORY OF RECURRENT STUTTERING AND RECOVERY IN TWENTY-FIVE YEAR OLD POST GRADUATE COLLEGE STUDENT

H. J. HELTMAN, Chairman

School of Speech and Dramatic Art

Syracuse University

J. C. (boy), seven year old stutterer, tonic spasms of the muscles of the jaw, with a right side downward thrust, mouth open, high, weak vocal tone, no inflection. Had four months instruction beginning February, 1923, two half-hour lessons weekly. No apparant improvement June 1st. Boy went away to camp for the summer. No professional or casual contact with this case again until 1928 when he was 12 years old.

I saw him standing on a corner and engaged him in conversation I discovered there was no trace of his former stuttering disorder. I did not see him again until he was a senior in high school in 1933, when he called on me professionally to get aid for the disorder which had again made its appearance during the latter half of his third year, and remained during the first half of his senior year. Both clonic and tonic spasms of the musculature of the neck and jaw as well as the tongue marked the extreme symptoms. His excuse for returning, however, was to receive some coaching for an oratorical contest in which he was scheduled to take part. He had memorized a speech that had obviously been coached in a rather bombastic style of delivery by some person who was considerably older. In reciting his speech to me he was able to get through it without a single stuttering symptom. In the meantime, every attempt at conversation was a failure because of the seriousness of the stuttering symptoms. In this particular contest he succeeded in winning the city championship, the state championship, and was given third place in the national in Washington. The stuttering remained. That was the last contact I had with him until two weeks ago, when I saw him again in a chance meeting in the city. We visited for ten minutes, during which time he showed no trace of stuttering symptoms. His voice is flexible and pleasant. He is well-poised and speaks with a rhythm in his speech that is as natural as one would find in the cultured speech of anyone who has never been afflicted with stuttering. During the conversation he told me that after he had seen me the last time, and incidentally, after the series of contests, he had gone to college to take his bachelor's degree, still stuttering very seriously. In college, however, he became interested in debating, entered into the activity wholeheartedly and, as he stated it, never had any trouble after that. It will be four years next June since he completed his bachelor's work.

In this connection may I call attention to a statement I made ten years ago in my mimeographed notes for my beginning course in Speech Re-education: "For those cases for which we have specific records of recovery with no relapses over periods of eight years or longer, there appears to have been in every case, after the treatment, participation in speech activities such as debating and contest speaking while in school, and considerable group and platform speaking for those who have finished school and have gone out into adult life." This, I believe, is the cue to the solution of the perseveration problem in the successful treatment of stuttering.

PHENOMENA RESEMBLING APHASIA, AGNOSIA, AND APRAXIA IN MENTALLY DEFECTIVE CHILDREN AND ADOLESCENTS[1]

DR. AUGUSTA JELLINEK
Central Institute for the Deaf,
St. Louis

Abnormalities in the development of mentally defective children appear both in their general behavior and in insufficiencies of particular capacities and functions. On the whole, persons whose intelligence is subnormal are not capable of orienting themselves in the world in the same way that normal people do. They cannot adequately adjust to changed circumstances, nor acquire, reproduce, and use information like others. It seems as if they were not able to understand the phenomena around them, nor to react to these phenomena normally. Detailed examination of individual cases usually shows that visual and auditory perception, and probably also cutaneous perception is not so exact, complete, constant, nor dependable as in normal persons. This pathological condition is not to be designated as agnosia, because agnosia, by definition means a *loss* of gnostic capacities. Since these capacities were never fully developed in the feeble minded, they could not very well be lost. It may be stated, however, that these defects in perception are of a type also found in the agnosias, and that they can probably be traced to developmental abnormalities of certain parts of the brain. Each case must be examined individually to determine whether these abnormalities of the brain are due to birth injuries, to inherited factors, endocrine deficiency, or other illness.

In most mentally defective children the practical functions, i. e., the realm of action, has suffered along with the gnostic. These children often show poor co-ordination of their movements; they are especially awkward in using tools, in writing, drawing, etc., and even in walking, eating, and dressing. Similar phenomena are found in patients with apraxia, following brain injuries. We may, therefore, consider the behavior of the mentally defective as related to the apractic type. The etiology is just as variable as in perceptual and gnostic disturbances.

The following discussion will attempt to pick out from the general concept of feeble mindedness those phenomena which, in their total effect, are most important in conditioning the abnormal behavior of the child. My therapeutic measures, in these cases, were directed, on the one hand toward the training of perceptual and gnostic faculties, and on the other, toward the improvement of the practical capacities. On the basis of the similarities pointed out above, I

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employed the same methods of speech correction in working with feeble minded children and adolescents, which I used in treating cases of aphasia or agnosia. By these methods I succeeded in many cases in raising the level of intelligence and improving the general behavior of these children to a marked degree.

The observations described below were made on children and adolescents sent to me for speech correction by neurologists and by the "Instituto Biotypologico" in Rome. I also carried on speech correction work with similar cases in the public schools for abnormal children. It would seem to be significant that abnormalities of the endocrine system were present in almost all of these cases. Most of them were under hormone therapy during the whole period that I observed them.

It is evident that feeble minded and abnormal children cannot develop a true picture of the external world so long as their perceptual and gnostic functions are imperfect. For this reason they cannot be taught in the same way that normal children are; nor can they profit in the same way from their instruction. This is the basic idea in therapeutic pedagogy. Since language and the comprehension of language are of fundamental importance in the development of intelligence, we must pay particular attention to the aphasic type of speech disturbance which is so frequent in the feeble minded. So long as these children are unable to speak correctly and understand properly, they cannot build up normal social relationships with other people. Since there is a very close connection between motility and intelligence, it is clear that motor defects may have a detrimental effect upon mental progress. The estrangement from the real world, the difficulties in making adjustments of all kinds, which we often observe in these children, may very well originate from the vague and shifting impressions of the external world, which are characteristic of the agnosias. It is quite conceivable that the symptoms of pathological anxiety, frequently found in such children, have a similar origin. The treatment, therefore, consists in speech therapy, in improvement of the gnostic functions so far as possible, in training of the motor system through gymnastics, manual training, etc., and in measures directed toward relieving the apractic uncertainty and insecurity of the patient. The whole program must be supplemented by general psychotherapy and therapeutic pedagogy.

Three cases will illustrate this hypothesis.

Case 1: S., a sixteen year old French boy of the asthenic type. S. was very tall for his age, had a dysplastic malformation of the thorax, and a high arched palate.

The development of the primary and secondary sex characteristics was retarded. He had been under hormone therapy for a year, during which time he had grown a great deal and puberty had begun. He had many vagotonic stigmata, blushed and paled frequently, and often had damp, cold hands.

His speech showed various abnormalities. When he was excited, he spoke extraordinarily quickly, with a decided tendency to logorrhea and with a slight degree of flight of ideas. It was difficult to stop him when he had once started in this way, and he would talk on for several minutes without interruption, in a high pitched voice, and with constantly increasing speed. At such times it was scarcely possible to understand him. When he was calm, he spoke fluently with good sentence structure, normal inflection and speed. He spoke correct French and could make use of a number of idiomatic phrases. But even at its best his speech was not easy to understand, because his articulation was unclear and he frequently lapsed into paraphasia. "S," "sh," and "z" were articulated laterally. His vocabulary was small. His voice had not yet changed and was still high and squeaky like a child's. His comprehension of language was very deficient; sometimes he was incapable of understanding a question and asked several times to have it repeated. His deficiency in understanding was also evident in the way he carried out commands. The perceptual disturbance showed itself most clearly when S. was asked to repeat words or sentences, and especially groups of nonsense syllables, if the mouth of the speaker was hidden. He could not repeat more than three nonsense syllables correctly and did not even make an attempt, when he was given five or six (a typical symptom in sensory aphasics). On the other hand he sang from memory correctly. With simple melodies he could keep in time with the music and could recognize melodies which were played on the piano or whistled. He usually designated noises correctly. Thus his perceptual disturbances resembled those encountered in sensory aphasics.

His parents complained that he had never succeeded to read fluently and could not understand at all what he did read. Even when he read the single words correctly he mostly did not know what they meant. At the beginning of the treatment the boy read softly and quite rapidly with frequent hesitation, pauses, and paralexia. Often he stopped after a few lines and said he couldn't go on. When he was asked what he had read, he could only tell approximately what it was all about, but could not give back the full content. He said himself that he could not read correctly. We find similar phenomena in patients with alexia following brain injuries.

S. could write easily with fairly good spelling, but only under certain conditions. He wrote approximately correctly from dictation, made many mistakes when he copied, and could not be persuaded to write spontaneously at all. When he wrote from dictation for a fairly long time, the number of mistakes increased rapidly.

He was entirely incapable of making even a rough drawing of an object from a model. When he was asked to draw a tree, a face, a flower, a fork, etc., he could accomplish nothing but a meaningless scrawl. He could not even make the primitive sketches of these objects which normal children of four and five can produce.

He could read and write numbers and could even pose fairly difficult arithmetical problems. But he had no comprehension of the operations involved in the problems, and quickly became lost in a muddle of very rapid speech, full of senseless circumlocations and perseverations.

When I first saw the boy, he was wearing glasses prescribed for very severe astigmatism, and he maintained that he could not see without them. The uncertainty of his movements made me suspect that he was not able to orient himself adequately through vision. Upon examining him for visual agnosia, a significant deficiency in the ability to recognize pictures became evident. At the beginning of the treatment he was also uncertain about the names of colors. I therefore supposed that his visual difficulties were central rather than peripheral. When I had him remove his glasses he was able to see just as well without them. His behavior was like that of a patient with visual agnosia. When he was asked to describe pictures he gave the most fantastic and variable answers.

In his motor behavior also S. showed various abnormalities. His movements were slow, awkward, and disproportionate. He could not be persuaded to go out alone, and always clutched his attendant's arm tightly. He took very small, cautious steps, and was afraid to go up or down stairs. If he could not hold on to anyone, he grasped the bannister and became quite pale with anxiety. When I first saw him, he was not able to dress or undress alone. His food was cut for him. In fact, he did not do anything by himself that was at all complex. His posture too was abnormal; he walked with his shoulders hunched up, his head down, and his eyes squinting; frequently he grimaced.

The whole behavior of the child was analogous to the type which is found in apractic patients. He showed the characteristic hesitancy, uncertainty, and awkwardness of movement, as well as the anxiety about doing things by himself.

His intelligence might be compared to that of a six or seven year old, but in some things he did not even reach this level. He did not always seem to be oriented as to time and place. His concepts of distance and of periods of time were incorrect. He never occupied himself alone, but was almost always with other people. This extremely dependent behavior was in part the consequence of his upbringing. For several years he had been in a boarding school. His parents became dissatisfied with the school, and, during the year preceding my acquaintance with him, his mother had taken care of him and taught him with the most solicitous devotion.

Emotionally he was not so retarded as he was mentally. But he was extremely excitable and took violent likes and dislikes to people. One of the most striking things about him was his pathological anxiety. He was afraid of everything unknown, was terrified by big signs in the street, was afraid of the dark, and was decidedly anxious with regard to unaccustomed motor activities, such as playing ball, or gymnastics. He was equally afraid of walking by himself in the

streets. This attitude was most extreme at railroad stations and trains. There he became violently agitated, flushed and paled by turns, began to tremble, and could not be budged even by force. He frequently expressed this fear of railroad trains in conversation.

Similar attacks of fear or rage, in which he trembled and showed extreme rigidity with violent vasomotor reactions, appeared also at other times if he became excited. The psychiatrist who was consulted expressed the opinion that these symptoms were equivalents for epileptoid attacks. He characterized the whole behavior of the boy as hypomanic.

S. was sent to me by an internist (Prof. N. Pende) for speech correction and therapeutic pedagogy. For over a year he had been receiving hormone therapy, which was continued over the whole period during which I worked with him. Under the influence of this therapy his behavior changed markedly. Hypophyseal and testicular preparations in different proportions were prescribed over three month periods. It was usually impossible, however, to treat S. for this length of time with the same medication, for after five or six weeks he suffered such violent agitation that the treatment had to be changed, and frequently sedatives had to be given. Under the influence of the hormone preparations his physical development progressed very quickly and approached the normal level of his age.

My task consisted not only in teaching the boy to speak, to read, and to write, but also in general personality training, so that he might, in so far as possible, become independent and capable of further education.

The parents, who were first cousins, were very gifted and cultivated people. They said the boy had had a normal birth, had never been ill; and they were at a loss to explain his abnormal development. His younger sister was an intelligent, well developed child.

There was no reason to suppose that the child had had a brain injury, unless it had occurred at birth without the knowledge of the parents. S. impressed the observer, however, as a case of organic brain pathology.

I treated his deficiency in the understanding of speech in the same way in which I treated sensory aphasics. I had him repeat nonsense syllables after me, also short, and then longer sentences, keeping my mouth hidden. I showed him pictures which I described, and then asked him to find certain objects in them. I gave him a number of pictures and asked him to pick out the one which I designated, always keeping my mouth covered. These tasks were very difficult for him because of his visual defects.

Great stress was laid on the practise of motor activities. His apractic awkwardness was improved by showing him how to do things and having him repeat certain actions over and over again. He was given instruction in gymnastics; later he was taught to swim, to catch a ball, and then to play football. He was urged to carry out the routines of every day life in so far as possible by himself. He

was taught to control the movements of his hands and fingers by picking up small objects such as matches, peas, stamps, etc. He learned to string beads, to model in clay, to cut figures out of paper, etc. Thus his apractic symptoms gradually improved. And this improvement alone helped to relieve his pathological anxiety.

During the first weeks it was impossible to treat his speech. His ability to concentrate and to pay attention was slight. For months he was unable to tell the difference between his own speech and the sounds, words, and sentences, which were spoken to him, for his perception of acoustic phenomena was not yet normally developed.

In order to train his visual perception, we practiced systematically distinguishing colors and forms. I used books especially designed for this purpose, in which incomplete drawings were to be completed, etc. We also made use of a number of games for the same purpose. In the course of several months his visual perception showed marked improvement. He described pictures correctly and was able, when asked, to choose the right one out of a fairly large number. He was constantly urged to draw and to paint, and within a few months he reached the level of a seven or eight year old in these capacities.

In teaching him to read and write, I used a method suggested by *Froeschels* in the treatment of alexia. The names of certain colors were written in the corresponding color, for example—"red" in red, "blue" in blue, "violet" in violet. Although not all the letters of the alphabet appear in these words, nevertheless most of them appear in the names of the colors. Thus this method is of great assistance to the alectic patient, for he knows that the letters "r" "e" "d," written in red, must appear in the word "red," and he need not therefore choose among 26 similar symbols, but only among three. The same holds for blue, etc. In the beginning I had him write the letters with a brush in water colors in order to make the experience of color more vivid.

General educational methods of personality training were employed with the idea of making the boy more independent, of accustoming him to spontaneous work, and of instructing him so far as possible in such a way that his education could be continued The program of his daily life was planned in detail, and carried out under the supervision of two trained assistants.

As a result of all these measures S. made marked progress and began to approach the normal level of his age group. He began to read and write spontaneously. He was even able to read the names of stations from a moving train. He liked looking at illustrated books and magazines. After several months he was able to listen with enjoyment to whole concerts, and was able to follow a moving picture, even though he could not understand the language, since it was in Italian. He played ball and football with other boys and had learned to walk and run alone on the street. His ability to orient himself had decidedly improved, and he often came a distance

of several blocks by himself to his lesson. He was able to make small purchases; he recognized the food products in the markets, and knew approximately what they cost. He had attained a secure relationship to reality, which had previously been entirely lacking.

After some months his comprehension of speech had decidedly improved and his vocabulary was considerably larger. When he did not understand a word, he asked what it meant. He even began to understand the Italian which his companions spoke, and often answered them quite correctly.

His paraphasia had decreased greatly. The tempo of his speech was slower, and his articulation clearer. His speech was, in fact, now quite understandable. At this stage I began to treat the mutation disturbances of his voice.

Since his apraxia was greatly meliorated, S. was now able to practise jaw movements which helped him to lower the pitch of his voice. The treatment of the lateral sigmatism was very difficult, however, for he was still unable to differentiate between the sound of the correct "s" and that of the lateral sound.

S.'s memory was now greatly improved. He was able to give back the content of short passages or stories not only immediately after reading them, but also on the next day. In order to develop his style, I used a French exercise book for ten year old children, in which missing words are to be filled in in short sentences. After four months S. was able to fill in the right words and usually chose the correct grammatical form.

Arithmetic caused him the greatest difficulty of all. Even after months of effort he was unable to add, subtract, multiply, or divide numbers of two or three digits without mistakes.

His emotional behavior had become more balanced. He appeared to have overcome his pathological anxiety almost completely. I believe that his visual agnosia had been one of the chief roots of this anxiety. The objects around him evidently did not have any constant form for him, but continually changed in appearance, so that he could never really become acquainted with them, and always found himself face to face with something unknown. He also had a tendency to experience everything as if it were animated. When I once asked him why he was so afraid of trains, he said, "He looks at me, he has such big eyes." Thus it seemed to me that he experienced the train as a threatening monster which was approaching him. Once when we were travelling on the railroad and the train slowed up at a station, S. said, "The poor thing, he is tired." His fear of the dark was undoubtedly intensified by the fact that in the dark he was quite incapable of recognizing anything. As his visual perception improved, his anxiety decreased. It seems permissible, therefore, to postulate a causal connection.

After ten months of treatment S. returned to France because of the uncertain political conditions. There he was sent to an agricultural school, where he took part fairly well in the general activities.

His whole behavior was not yet normal, but very decidedly improved. The hormone therapy had been continued during the whole period of the speech correction work and had lasted altogether three years. Since the significant change in his behavior, however, appeared first during the speech therapy, it seems reasonable to attribute his improvement in large part to this treatment.

Case 2: B., a twelve year old feeble minded Spanish girl. This child too was sent to me by her internist (Prof. N. Pende). She also was under hormone therapy during the whole period of the treatment. The girl was very pretty, but abnormally fat, and still had a very childlike figure. At the beginning of the treatment she seemed very dull. Her movements were slow and awkward. She showed almost no change in facial expression and was easily frightened by unaccustomed stimuli. She spoke scarcely at all, understood little and could neither read nor write. She evidently enjoyed music very much and her facial expression changed decidedly when someone played to her. She sang very nicely, carried a tune and kept time correctly. Sometimes she could even sing the words of a song. Although her movements were usually very awkward, she danced quite rhythmically and with much more relaxed motor activity.

The child had grown up in a polyglot environment. Her parents spoke Spanish and Portuguese. At an early age she had had an English nurse for a year. Her next nurse spoke only Spanish. Then she moved with her family to France. I spoke to her in French, but at home, the child heard, besides French, Spanish from her nurse and Italian from the servants. When I first saw her, she could understand a little of all these languages but none of them perfectly.

B. shows a decided liking for pretty things, especially new clothes. In spite of her awkwardness, she had the good manners of a little lady. Her spontaneous speech was limited to a few Spanish words and loud cries when she was pleased about something. At the beginning of the treatment, her behavior was on the whole rigid, like an automaton, and she could scarcely ever be brought to do anything spontaneously.

She showed a striking echolalia, very marked and very exact. At the beginning she repeated almost everything that she heard, even when it was spoken in a language which she did not know. If I asked her something, she did not answer, but repeated the question with the same inflection which I had used. This sort of behavior corresponds to a symptom frequently encountered in sensory aphasia. B. showed this echolalia, however, only when she could be roused from her lethargy and forced to pay attention.

There seemed to be a deficiency in the inhibitory mechanisms of her acoustic centers. A polyglot environment sometimes delays or hinders the development of language even in a normal child. Little B., with her extraordinarily receptive speech centers, had probably perceived elements of different languages simultaneously without being able to keep them separate in her mind, and without being able

to form definite connections between words and objects. It seems to me that in her case this was one of the chief reasons why, at the age of twelve, she was still unable to express herself in speech. B. was undoubtedly feeble minded and abnormal in many respects. But one must take into consideration the fact that she could not learn and teach herself by asking questions like other children. Thus the faculties which she did possess could not be developed. In order to call her attention to the motor-kinesthetic aspect of speech, I used life sized pictures of a face with conventionalized lips, drawn in the position for articulating a particular sound. As I showed her each picture I imitated the position of the mouth and made the proper sound. After a time she knew the pictures so well that upon seeing them, she could make the sound which each one represented. When she had accomplished this, I added letters to the pictures, putting with each picture the letter corresponding to the position of the lips. In this way the visual image of the letter was brought into relationship with the corresponding motor pattern and with the sound. After some months B. learned by this method to use some of the letters of the alphabet. (Method by Fröschels for the treatment of apasics).

In order to establish a connection between the sound of a word and the object, I showed her simple pictures which she was to name and later also to describe in short sentences. B., with her typical echolalia, repeated the words immediately. It was a long time, however, before she could independently designate pictures with the words, which she had imitated so easily. This method of showing her pictures also served to hold her attention and to train her capacity for concentration.

She did not seem to have any symptoms of visual agnosia.

In teaching her to read I used the same picture of the face with conventionalized lips, mentioned above. Since it was so difficult to hold her attention I used letters about three inches high, made out of red or yellow cardboard which she was to put on the corresponding cards. After months of effort she learned a few letters and began to read words.

As a preliminary to teaching her to write, I had her fill in the outlines of various figures with colored crayon (Montessori method). Then I gave her large colored wooden letters, the outlines of which she was to trace in pencil. She did not, however, learn to copy letters or to write spontaneously.

Her motor activity was trained both by rhythmic gymnastics, ball games, etc., and also by various manual occupations. She learned to dress herself alone, to do housework, embroider, etc.

All of these efforts resulted in this case also in a significant improvement in behavior. B. was livelier; her face showed animation; and her eyes became expressive. She enjoyed the company of other children and began to play with them. After some months she finally began to speak spontaneously. She asked for certain objects,

related her little experiences, and seemed to like to listen when people talked to her. To my surprise, her first rudimentary expressions were polyglot. To me she spoke French; to her nurse, Spanish; and to the cook, she spoke the few Italian words that she had been able to pick up. In Spanish, however, she had the largest vocabulary and spoke most fluently, due perhaps to the impression made upon her by that language in childhood. After a year in Rome she knew enough Italian to be able to carry out simple commands. Unfortunately I was not able to keep her in an environment in which only one language was spoken. The treatment lasted about 10 months.

The whole behavior of the child had become more independent. She frequently occupied herself alone, and her previous anxiety had greatly decreased. Above all, she was now at a stage in which she could profit from further instruction.

Case 3: T., a five year old Italian girl. T. was in a home for feeble minded children and had been classified as an imbecile by the consulting neurologist. Her body was well formed and she was very pretty. Her motor activity was normal. Whenever she heard music she began to dance spontaneously. She could eat, run, jump, and climb with perfect ease, and had good manual dexterity. Frequently, however, she became perfectly rigid for a short time without any apparent reason. During these periods she stared straight ahead with an expressionless face, and made no response to any attempt to rouse her. At times when she was particularly excited, another strange symptom appeared; she rocked back and forth rhythmically for hours at a stretch, singing the same few notes over and over again.

When I first saw her she did not speak at all and could not even imitate words. She could, however, imitate with perfect accuracy any action which struck her fancy. She had developed a kind of autistic language-patter, which was quite incomprehensible, and in which one could not distinguish any words. But the intonation was so similar to that of normal speech, that from a distance one might think that she was really talking. She could imitate the inflection and timbre of foreign languages in the same way. It was impossible, however, to induce her to repeat single words or short sentences correctly. She seemed to have some understanding of language. For hours she would sing and talk to herself in her own patter. She was quite capable of carrying a tune correctly with good rhythm.

A year later I began to treat the child. She still had the habit of imitating people's actions. If I touched my right cheek with my right hand, she did the same thing to herself. If I touched her right hand, she did not touch mine, but touched her own right hand with her left. If I removed an object from the table and put it back, she repeated the action with the same object. She did not speak spontaneously, but her comprehension of speech was greater, and her autistic individual speech had disappeared. She showed very marked and exact echolalia and repeated sentences without mistakes. She could carry out simple commands, but was unable to understand a description of a picture. When she was asked to point out a flower,

or an animal in a picture, she was quite at a loss and pointed indiscriminately at various parts. She was incapable of choosing a designated object out of a number presented to her. Her symptoms were similar to those which are frequently found in cases of sensory aphasia. She had a good musical memory. She could sing a number of melodies perfectly correctly with good rhythm and often with words. She still danced rhythmically when she heard the radio.

The child had learned a number of the letters of the alphabet, could copy correctly, but could not write spontaneously, nor from dictation. She could read letters, was very uncertain about words, and had no idea what a printed word meant.

Her attention fatigued easily and was very difficult to hold. At times it was impossible to establish any contact with her. She withdrew into herself and refused to respond to any kind of treatment. I treated her in the same way as the previous case, like a case of sensory apasia.

After weeks of effort T. learned that certain words belonged with certain objects. From this time on she made marked progress. She learned to name a number of objects independently and to identify the objects when she heard their names.

Since the School for Feebleminded had charge of T.'s general training, I had to limit myself in this case to speech therapy. Nevertheless in the course of several months the child not only began to speak and to enlarge her comprehension of speech, but she also began to approach a more normal level in her general behavior. In particular, her tendency to imitate the actions of other people had greatly diminished. Her attention was more constant and she was in closer contact with her environment. This would seem to be due in large part to her increased understanding of language. She was now able to learn more easily about what was going on around her. It would seem also that her ability to express herself in speech, even though slight, gave her an outlet for her desire for motor activity. In any case, with the ability to speak she had become less restless. With T., as well as with the other children, the recovery from a condition, similar to aphasia, seems to have resulted in a higher level of intelligence.

Many other cases could be cited which were treated with equal success, on similar principles.

In the public schools for abnormal children in Rome, about one-third of the cases were afflicted with more or less serious defects in speaking, reading, and writing. I treated these children with the usual speech-corrective methods of the Vienna school. I shall mention particularly those cases of reading and writing defects with which the public school teachers were unable to make any progress. I treated the children as if I were dealing with aphasic and alectic disturbances. I employed the methods described above with colored letters and the life sized pictures of the face.

Idiots were not accepted in these schools, but with the imbeciles and subnormal children, whom I treated, I always succeeded in

teaching them to read. I always combined writing with reading and tried to improve the ability to write by exercising the hands. For this purpose I had them pick up small objects, model in clay, draw, paint, etc. It seems reasonable to suppose that this sort of exercise tended toward a general improvement of manual dexterity.

The progress of a child in school depends, of course, largely on his ability to read and write. These two capacities also significantly influence the development of memory and attention. We may therefore expect that the successful treatment of defects in reading and writing will raise the level of intelligence.

The cases quoted illustrate sufficiently our theory, that abnormalities in intelligence, emotion, and social behavior can be decidedly improved by meliorating the fundamental defects, similar to apraxia, agnosia, and aphasia, from which almost all of these persons suffer. In this paper we have been concerned only with children and adolescents, who because of these defects had never been able to establish normal relationships with their environment.

Every individual who deviates from the norm calls forth from his environment an abnormal reaction of one kind or another. He may encounter over-indulgence and exaggerated assistance, which restricts his already reduced activity. He may meet with cruelty and derision, especially on the part of his contemporaries. His parents and teachers may show no interest in him, because he responds so slowly and imperfectly to instruction. Thus his constitutional defects are intensified by the reactions produced in the environment. Because of these factors it is very difficult to ascertain what part of the behavior of an abnormal child is due to an irreparable deficiency and how much might be improved by training.

It seems reasonable therefore to attempt a therapeutic approach to such cases both because of the possible practical results and also on account of the value to research. We may justifiably hope that a young organism will be able to develop normally if it can make complete normal contacts with the outside world. The best methods of approaching the problem seem to be those which are therapeutically effective in aphasia, apraxia and agnosia. It is highly desirable that these methods be tested on a larger number of cases than I had at my disposal.

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American Speech Correction Association

Membership List 1941

American Speech Correction Association

The American Speech Correction Association was organized in New York City in December, 1925, at the annual convention of the National Association of Teachers of Speech with which this organization has always been affiliated. Its original name, the American Academy of Speech Correction, was changed to the American Society for the Study of Disorders of Speech in 1927, and the present name was acquired in December, 1934.

The purposes of the American Speech Correction Association as set forth in its Constitution are:

1. To stimulate among educators, physicians, and others of the general public a deeper, more intelligent interest in problems of speech correction.
2. To raise as rapidly as possible existing standards of practice among workers in the field of speech correction.
3. To secure public recognition of the practice of speech correction as an organized *profession.*
4. To furnish this new profession with responsible and authoritative leadership.
5. To make this leadership generally respected by our good works, i. e., by our scholarly research work, publicity work, and administrative skill.
6. To make membership in our organization a coveted recognition of merit and in this way to furnish workers in the field of speech correction with a powerful incentive to greater achievements.

The Association is composed of Associates and Fellows. Any reputable speech correctionist with a college education or its equivalent is eligible for election as an Associate.

A Fellow is usually a pioneer or leader in the field of speech correction who has done eminent research work in speech correction and obtained the doctor's degree.

1941 LIST OF MEMBERS

The following alphabetical list of members of the Association gives the title, degree, professional rank, and address of every Fellow and Associate whose 1941 membership dues were paid on February 1, 1941, in accordance with Section 4 of Article III of the Constitution as amended at the 1938 Cleveland Convention. This is not a complete list of members.

NOTE.—In the following list the names of the Associates are indicated by (A) and the names of Fellows are indicated by (F).

Most of the abbreviations used are standard and self-explanatory The less obvious ones are as follows: correction—cor., improvement—imp., teacher—T.

Teachers teach in the city where their address is listed unless otherwise noted.

In listing institutions which have granted degrees, all state universities have been designated simply by the abbreviation of the state.

(A) ADAMS, MINNIE HARRIS, A.B. (Radford State Teachers) 1935, A.M. (Iowa) 1940. T., Speech Cor., Pub. Schools, 209 Lansing Ave., Lynchburg, Va.

(A) AINSWORTH, STANLEY H., A.B. (Mich. State Nor.) 1933, A.M. (Iowa) 1937. Sup. of Speech Cor., Coleraine, Minn.

(A) AITCHISON, ADAM MORTON GLASS, A.B. (Ripon Coll.) 1926, A.M. (T. Coll., Columbia) 1939. Instr. in Speech at Columbia; Tutor in Speech at Queens Coll., 65-30 Kissena Blvd., Flushing, N. Y.

(A) AMIDON, HILDA FARNUM, (Mrs.), S.B. in Ed. (N. Y. Univ.) 1935. Pub. Sch. Sup. of Speech Cor. and Lip Reading, 282A Sigourney St., Hartford, Conn.

(A) ANDERSON, DOROTHY IOLA, A.B. (Iowa) 1928, A.M. (ibid) 1931. Instr. in Speech, Univ. of Ill. 603 W. Green St., Urbana, Ill.

(A) ANDERSON, MARGARET LOUISE, A.B. (Knox) 1917, A.M. (Univ. Kans.) 1925. Asst. Prof. of Speech, Univ. of Kans. 1126 Louisiana St., Lawrence, Kans.

(A) ANSBERRY, MERLE, A.B. (Univ. Calif.) 1929, A.M. (ibid) 1931, Ph.D. (Wis.) 1937. Dir. of Speech Clin., Instr. in Pub. Speaking, Univ. of Virginia. Swiss Village, Rt. 1, Charlottesville, Va.

(A) AREY, MABEL-LOUISE, B.L.I. (Emerson) 1926, A.M. (Pa. State) 1937. Clinician of Remedial Reading at Dr. S. T. Orton's Clin. Barbizon-Plaza Hotel, West 58th St., New York City.

(A) ARNOLD, GENEVIEVE, A.B. (Minn.) 1934, A.M. (ibid) 1936. Pub. Sch. T. of Speech Cor. 607 W. Maple St., Austin, Minn.

(A) ATHERTON, GRACE WILLIAMS (Mrs.). In charge of Speech Cor., Washington, D. C., Pub. Sch.; Dir. of Speech Cor. Clinics, Wilson T. Coll. and Episcopal Eye, Ear and Throat Hosp., Lecturer, Wilson Teachers College, Washington. 9300 Conn. Ave., Chevy Chase, Md.

(A) ATTENBOROUGH, ELIZABETH MARIE, B.S. (Rutgers) 1928. T. Speech Cor., Pub. Sch. 319 Westfield Ave., Elizabeth, New Jersey.

(A) BACKUS, OLLIE L., A.B. (Mich.) 1929, A.M. (ibid) 1930, Ph.D. (Wis.) 1933. Chairman, Dept. of Speech, State T. Coll., Slippery Rock, Pa.

(A) BAKER, MAE HANNAH, B.L.I. (Emerson) 1921, A.B. (Keuka) 1927, A.M. (Columbia) 1928. Prof. of Speech and Drama and Speech Pathology, Keuka Coll., Keuka Pk, N. Y.

(A) BAKES, FRANK P., A.B. (Ohio State) 1924, A.M. (ibid) 1927, Ph.D. (Iowa) 1938. Psychol., Ohio Bureau of Juvenile Research, 2280 W. Broad St., Columbus, Ohio.

(A) BALL, MARIE AGNES, S.B. (Univ. Rochester) 1929, A.M. (ibid) 1932. Instr. in Speech, Ext. Div., Univ. of Rochester; Pub. Sch. Speech Specialist. 145 Seneca Pkwy., Rochester, N. Y.

(A) BANGS, JACK L., A.B. (Univ. Wash.) 1939. Dir. of Speech Clin., Instr. Speech Path. and Cor., Univ. of Oregon. 1418 E. 23, Eugene, Ore.

(A) BARNETT, ROBERTA, A.B. (Univ. Mo.) 1923, A.M. (Columbia Univ.) 1929. Dir. of Speech Clin. and Head of Speech Dept., State T. Coll., E. Stroudsburgh, Pa.

(A) BARRITT, VIRGIL CROOK (Mrs.), A.B. (Kansas Univ.) 1936. Instr. in Speech Cor., Wichita Univ. 1337 No. Vassar, Wichita, Kansas.

(A) BELL, ALTA REBECCA, A.B. (Ohio Wesleyan) 1918, A.M. (T. Coll., Columbia) 1927. Pub. Sch. T. of Speech Cor., 720 Bierman St., York, Pa.

(F) BENDER, JAMES F., S.B. (Columbia) 1928, C.P. (N. Y. S.) 1931, Ph.D. (Columbia) 1939. Asst. Prof. of Speech, Queens Coll., 65-30 Kissena Blvd., Flushing, N. Y.

(A) BERGQUIST, RUTH WHITTAKER (Mrs. Clarence W. Bergquist), A.B. (Malcalester) 1924. Pub. Sch. T. of Speech Cor., St. Paul. Box 168, Chicago City, Minnesota.

(A) BERRY, MILDRED FREGURG (Mrs.), A.B. (Iowa) 1922, A.M. (ibid) 1925, Ph.D. (Wis.) 1937. Prof. of Speech, Rockford Coll., Rockford, Ill.

(A) BERWICK, NAOMI HUNT (Mrs.), A.B. (Hardin-Simmons) 1929, A.M. (Iowa) 1939. Private Studio of Speech Cor., Box 296, Winters, Texas.

(A) BISHOP, JEANNE H. (Mrs. Robert S. Fisk).

(A) BISSELL, VELMA L. (Mrs. Hiser).

(A) BLACK, JOHN WILSON, A.B. (Wabash Coll.) 1927, A.M. (Iowa) 1930, Ph.D. (Iowa) 1935. Prof. of Speech, Kenyon College, Gambier, Ohio.

(A) BLANCHARD, DOROTHY LUCILE (Mrs. Thad A.), Pub. Sch. T. of Speech Cor., 312 Avalon Ave., Detroit, Mich.

(A) BLANCHETTE, ALVENA M., S.B. (Minn.) 1926. Pub. Sch. T. of Speech Cor. The Leomington, Minneapolis, Minn.

(F) BLANTON, SMILEY, S.B. (Vanderbilt) 1904, M.D. (Cornell) 1914. Diploma in Psychol. Med. (Royal Coll. of Phys. and Surgeons of London and England) 1923. Consultant in Psychiatry, Marble Collegiate Church, N. Y. City. 115 E. 61st St., New York City.

(A) BLAZEK, J. WALLACE, A.B. (Coe Coll.) 1934. T. of Pub. Speech, Speech Pathol. Clinician, Twp. H. S. 214 E. Church, Collinsville, Ill.

(A) BLOOMER, HENRY HARLAN, A.B. (Ill.) 1930, A.M. (Mich.) 1933, Ph.D. (ibid) 1935. Manager of Speech Clin. and Asst. Prof. of Speech, Univ. of Mich., Ann Arbor, Mich.

(F) BLUEMEL, CHARLES SIDNEY, A.M. (Colo) 1915, M.D. (ibid) 1916, M.R.C.S. (England) 1917, F.A.C.P., 1931. Physician, 550 Metropolitan Bldg., Denver, Colo.

(A) BOHANNON, DOROTHY ELIZABETH, B.S. (S. W. Mo. Techrs.) 1928, M.A. (Iowa) 1938. Teacher of Speech Cor., Pub. Schools. 1123 Jackson, Joplin, Mo.

(A) BOSLEY, ELIZABETH (Mrs. W. Elvis Bosley), A.B. (Friends Univ.) 1933, A.M. (Univ. Kansas) 1935. Instr. in Speech Clin., Flo Brown Mem. Lab., Univ. of Wichita. 823 Sedgwick, Wichita, Kansas.

(A) BRACKET, I. PARSONS, S.B. (Northwestern) 1938, A.M. (ibid) 1940. Asst. in Training of the Speaking Voice and Clin., Northwestern Univ. Sch. of Speech, Evanston, Ill.

(A) BRADLEY, RUTH JULIA, E.B. (Univ. Akron) 1926, A.M. (Columbia) 1932. Dir. of Speech Clin. and Asst. Prof. of English, Speech and Dramatics, State T. Coll. Wilimantic, Conn.

(A) BRIGHAM, FLEDA MARGARET, A.B. (Occidental Coll.) 1930, A.M. (S. Calif.) 1936, D.O. (L. A. Coll. Osteopathic Physicians and Surgeons) 1936. Jr. Attending Physicians, Neuro-Psychiatric Service, L. A. County Osteopathic Hosp.; Speech Cor., Private Practice at 1711 Monterey Rd., S. Pasadena, Calif.

(A) BRILTY, FRANCIS XAVIER, B.A. (Wis.) 1937, M.A. (Ohio State) 1940. Assistant in Voice Science and Phonetics Laboratories, Ohio State Univ. 1866 N. High St., Columbus, Ohio.

(A) BRITTON, HENRY CHASE, B.S. (Boston Univ.) 1934, M.A. (ibid) 1936. T. Speech Cor., Curry School. 115 Ashland St., Malden, Mass.

(A) BRONSTEIN, ARTHUR JORDAN, A.B. (Coll. City N. Y.) 1934, A.M. (Columbia) 1936. Instr. Dept. of Speech, Queens Coll., Flushing, New York. 7664 West 163rd St., New York City.

(A) BROWN, HELEN, S.B. (Boston Univ.) 1923, A.M. (Pittsburgh) 1929. Assoc. Prof. of Speech, Hampton Inst., Hampton, Va.

(F) BROWN, SPENCER F., A.B. (Shurtleff) 1933, A.M. (Iowa) 1935, Ph.D. (ibid) 1937. Instr. in Speech, Univ. of Minn., Minneapolis, Minn.

(A) BRYANT, MARIE BOLLINGER, (Mrs.), B.S. (Kansas State Teachers Coll.) 1922, A.M. (Columbia) 1926. T. of Speech Improvement and Cor., Jamaica High School, N. Y. C. 300 Merrick Road, Rockville Centre, N. Y.

(A) BRYNE, MAY EDITH, S.B. (Minn.) 1922. Pub. Sch. Dir. of Special Educ., 305 Court House, Minneapolis, Minn.

(F) BRYNGELSON, BRYNG, A.B. (Carleton) 1916, A.M. (Iowa) 1926, Ph.D. (ibid) 1931. Dir. of Speech Clin. and Assoc. Prof. of Speech, Speech Clin., Univ. of Minn., Minneapolis, Minn.

(A) BUCHOLZ, CLARENCE ARTHUR, A.B. (Baldwin-Wallace) 1923. B.D. (Drew Univ.) 1925, A.M. (T. Coll., Columbia) 1928, Instr. in Speech Pathol., Fenn Coll., Cleveland, Ohio.

(A) BURDIN, L. GRAY, S.B. (Butler Univ.) 1935, S.M. (ibid) 1937, A.M. (Northwestern Univ.) 1939. Instr. in Speech at Butler Univ. 839 W. 44th St., Indianapolis, Ind.

(F) BUSSE, ALVIN CLAYTON, A.B. (Macalester) 1920, A.M. (N. Y. Univ.) 1924. Assoc. Prof. of Speech, N. Y. Univ., University Hts., New York City.

(A) BUXTON, ROSE (Mrs.), B.A. (New York) 1936. Dir. of Speech Clin., Williamsburg Housing Project, Brooklyn. 110 Clara St., Brooklyn, N. Y.

(A) CABLE, W. ARTHUR, Ph.B. (Univ. Chicago) 1920, A.B. (Manchester Coll.) 1920, A.M. (Iowa) 1925. Dir. of Speech Clin. and Prof. of Speech, Univ. of Arizona. 524 E. Fourth St., Tucson, Ariz.

(A) CALVERT, RAYMOND R., M.D. (Ind.) 1927. Physician to Purdue Speech Clin., 314 N. 6th St., Lafayette, Ind.

(F) CAMP, PAULINE BEATRICE, Pub. Sch. Dir. of Child Guidance and Special Educ., Frost Woods, Madison, Wis.

(A) CARHART, RAYMOND THEODORE, A.B. (Dakota Wesleyan) 1932, A.M. (Northwestern Univ.) 1934, Ph.D. (ibid) 1936. Asst. Prof. of Speech Re-educ., School of Speech, Northwestern Univ., Evanston, Ill.

(F) CARLSON, EARL REINHOLD, A.B. (Minn.) 1921, S.M. (ibid) 1922, M.D. (Yale) 1931. Dir. Dept. of Rehabilitation of Motor Disorders, N. Y. Neurological Inst.; Dir., Sch. for Corrective Motor Educ., E. Hampton, N. Y., and Pompano, Fla. Lago del Mare, E. Hampton, L. I., New York.

(F) CARR, ANNA M., A.B. (Cornell) 1904, A.M. (Iowa) 1926. Dir. of Speech Clin. and T. of Speech, State Teachers' Coll., Milwaukee, Wis.

(A) CARRELL, JAMES A., A.B. (Neb. Wesleyan) 1927, A.M. (Northwestern) 1929, Ph.D. (ibid) 1936. Dir. of Speech Clin., Univ. of Wash., Seattle, Wash.

(A) CASEBOLT, JESSIE D., A.B. (S. F. State Coll.) 1935. Asst. Prof. of Speech, Drama, and Speech Imp., S. F. State Coll. 671 The Alameda, Berkeley, Calif.

(A) CASEY, MARY HELENA, B.L.I. (Emerson) 1925. Pub. Sch. T. of Oral Reading. 46 June St., Lowell, Mass.

(A) CASS, MARION THERESSA, A.B. (Univ. Nebraska) 1927, A.M. (T. Coll., Columbia) 1932. Sup. of Speech, Glen Cove High Sch. 56 Franklin Ave., Glen Cove, N. Y.

(A) CASWELL, ELIZABETH (Mrs. W. Elvis Bosley).

(F) CHAPIN, ALICE C., A.B. (Univ. Penn.) 1916, A.M. (S. Calif.) 1923. Prin. Speech Cor. Sch., Los Angeles City Schools; Instr. Ext. Div., Univ. of Calif. 610 N. Kenmore Ave., Los Angeles, Calif.

(A) CHAPMAN, MYFANWY ELIZABETH (Mrs. Kirt M. Chapman), S.B. (Minn.) 1937. Pub. Sch. T. of Speech Corr. 5344 Ewing Ave., S., Minneapolis, Minn.

(A) CLANCY, JOHN N., Ph.B. (Univ. Notre Dame) 1921, A.M. (Mich.) 1937. Dir., Shady Trails Nat. Speech Imp. Camp.; Admittance Officer and Clinician, Univ. of Mich. Speech Clinic, Ann Arbor, Mich.

(A) CLAPP, JEAN CHRISTINE, A.B. (DePauw Univ.) 1935. Asst. in Speech Re-educ., Northwestern Univ. 1637 Orrington Ave., Evanston, Ill.

(A) CLARK, RUTH MILLBURN, (Mrs.), A.B. (Univ. Utah) 1921, A.M. (ibid) 1935. Pub. Sch. T. of Speech Cor. 227 S. 13 East, Salt Lake City, Utah.

(A) COBLENTZ, IRVING, B.S. (Penn. State) 1938. Clinician, Speech Clinic, Pennsylvania State College. 532 West College Ave., State College, Pennsylvania.

(A) COCHRAN, VALMA DERINGTON, (Mrs. Fred Cochran, Jr.), A.B. (Wichita Univ.) 1937. Instr. in Corrective Speech. 1501 N. Waco, Wichita, Kansas.

(F) COTTON, JACK CHILTON, A.B. (Maryville) 1929, M.Sc. (Ohio State) 1930, Ph.D. (ibid) 1936. Dir. Speech Clin. Laboratories, Western Wash. Coll. of Educ. 1537 James St., Bellingham, Wash.

(A) CREWS, LOIS MCSLOY (Mrs. A. R. Crews), S.B. (Northwestern) 1929, S.M. (ibid) 1932. Instr. in Training of the Speaking Voice and Speech Re-educ., Northwestern Univ. 1935½ Orrington Ave., Evanston, Ill.

(A) CROSS, HILDRETH MARIE, A.B. (Asbury Coll.) 1922, A.M. (Mich.) 1927, Ph.D. (Iowa) 1933. Assoc. Prof. of Educ. and Psychol., Asbury Coll., Wilmore, Kentucky.

(A) DAMON, KENNETH F., A.B. (Wis.) 1922, A.M. (T. Coll., Columbia) 1927, Ph.D. (Columbia) 1933. Asst. Prof. of Public Speaking, City Coll. 17 Lexington Ave., New York City.

(A) DAMON, RUTH AIKMAN (Mrs.), A.B. (Kent State) 1921, A.M. (Northwestern) 1923. Asst. Prof. of Speech, Adelphi College, R. No. 1, Wadsworth, Ohio.

(A) DAVIES, NELLIE ELEANOR, Ph.B. (Wis.) 1931. Pub. Sch. T. of Speech Cor. 33½ Park St., Oshkosh, Wis.

(A) DAVIS, ANNETTA CAROLYN, A.B. (Wichita Univ.) 1938. Instr. in Clin., Wichita Univ., Flo Brown Mem. Lab., Wichita, Kansas.

(F) DAVIS, DOROTHY MAY, (Mrs. C. Tuthill).

(A) DAVIS, LEOTA MAUDEANE, A.B. (Univ. Okla.) 1938, A.M. (Wis.) 1939. Asst. in Speech, Purdue Univ. 525 State, W. Lafayette, Indiana.

(A) DAVISON, LOUISE DAVIS (Mrs. W. W. Davison), A.B. (Worthen) 1907. Co-Dir., Jr. League Sch. for Speech Cor.; Private Sch., 1780 N. Decatur Rd., Atlanta, Ga.

(A) DAWES, ROBERT GATES, A.B. (Swarthmore Coll.) 1939, A.M. (Columbia Univ.) 1932, Ed.D. (Temple Univ.) 1937. Dir. of Sch. of Dramatic Art, Ohio Univ., Athens, Ohio.

(A) DEMCHAK, AMY MARIE (Mrs. Jessie Demchak). Pub. Sch. T. of Speech Cor., 16524 Bentler Ave., Detroit, Mich.

(A) DENNISON, WILLIAM, Dir. of Speech Clin., Dennison School of Speech Correction. 543 Jarvis St., Toronto, Ontario.

(A) DERZAWETZ, MAURICE, S.B. (Franklin & Marshall Col.) 1936. Asst. Dept. of Psychology, Univ. of Iowa, Iowa City, Iowa.

(A) DIAMOND, MATTHEW, B.A. (New York) 1939, M.A. (ibid) 1940. Dir. of Speech Clin., The Educational Alliance, 197 East Broadway, New York, N. Y.

(F) DICK, ARTHUR, 1st. Lt., M.C., D.D.S. (Georgetown) 1931, M.D. (ibid) 1936. Station Hospital, Fort Meade, Md.

(A) DIEMER, MARY G., S.B. (Drake Univ.) 1930. Pub. Sch. Speech Therapist. P. O. Box 146, H. P. Station, Des Moines, Iowa.

(A) DILLER, GENEVIEVE MANN (Mrs.), B.S. (Millersville State Teachers Coll.) 1935, M.Ed. (Penn. State) 1940. T. Speech Cor., Pub. Schools. 527 N. Duke St., Lancaster, Pennsylvania.

(A) DOURIS, ELIZABETH FOOTE (Mrs. Patrick J. Douris), A.B. (Hunter), A.M. (Fordham). Co-chairman English and Speech Dept., New Utrecht H. S., Utrecht. Hotel Bossert, Brooklyn, N. Y.

(A) DOW, CLYDE WALTON, B.L.I. (Emerson) 1931, S.M. (Mass. State Coll.) 1937. Instr. in Pub. Speaking, Mass. State Coll. 116 Montague Rd., N. Amherst, Mass.

(A) DRUSHAL, J. GARBER, A.B. (Ashland Coll.) 1935, M.A. (Oho State) 1938. Clinician, Speech Clinic, Children's Hospital; Instr. of Speech, Capital University, Columbus, Ohio. 54½ S. Drexel Ave., Columbus, Ohio.

(A) DUKE, DARLENE (Mrs. Irvin F. Keeler).

(A) EGER, MARYAN EARL (Mrs.), A.B. (Univ. of Wichita) 1938. 1437 Kearsley Park Blvd., Flint, Michigan.

(A) ECKELMANN, DORATHY ANNE, S.B. in Ed. (S. E. Mo. T. Coll.) 1929, A.M. (Mo. Univ.) 1938. Sup. of Pub. Sch. Speech Clin. and T. of Pub. Speaking, Central H. S. 1225 Counsin St., Cape Girardeau, Mo.

(A) ELLENBERGER, MARTHA V., A.B. (Pittsburgh) 1925, A.M. (ibid) 1933. Pub. Sch. T. of Speech Cor., 551 Cypress Ave., Johnstown, Pa.

(A) ELLIOTT, LUCY A. Raleigh Apts., 3664 Washington St., St. Louis, Mo.

(A) ESPAILLAT, GRACE RUTHERFORD (Mrs.), T. Speech Cor., Pub. Schools, District of Columbia. The Chastleton, 16th and R. St. N. W., Washington, D. C.

(F) ESTABROOK, EUDORA PORTER, A.B. (Mich. State Nor. Coll.) 1929. Prin., Diamond and Congress Elem. Schs. 511 Ethel Ave., S. E., Grand Rapids, Mich.

(F) EVANS, MARSEE FRED, A.B. (Cornell Coll.) 1915, A.M. (ibid) 1917, B.D. (Drew) 1924, Ph.D. (Iowa) 1932. Prof. of Speech, Birmingham-Southern Coll., 1521 Eighth Ave., W., Birmingham, Ala.

(A) EWELL, ALICE MARIE, B.A. (Smith Coll.) 1938, M.A. (Columbia) 1940. Staff Psychologist, Speech Clinic, Purdue Univ. 525 State St., West Lafayette, Indiana.

(F) FAGAN, HELEN ROSE, S.B. (Washington Univ.) 1933, A.M. (Iowa) 1937. Asst. Dir. of Speech Clin., Central Inst. for the Deaf and Washington Univ. Coll. 818 S. Kingshighway, St. Louis, Mo.

(F) FAGAN, LEO BERNARD, A.B. (Creighton) 1925, A.M. (Iowa) 1927, Ph.D. (ibid) 1929. Psychologist and Speech Pathologist, St. Louis Psychiatric Child Guidance Clin. and Assoc. Prof. of Educ. Psychol., St. Louis Univ. 15 N. Grand Blvd., St. Louis, Mo.

(A) FAISON, MARTHA H., S.B. (T. Coll., Columbia) 1919, A.M. (ibid) 1920. T. of Speech, Lochland Sch., Geneva, N. Y.

(A) FARRELL, ELEANOR AGNES, S.B. (N. Y. State T. Coll.) 1934. Pub. Sch. Sup. of Speech Imp.; Hotel Chelsea, W. 23rd St., New York City.

(A) FIELDS, VICTOR A., S.B. (C. C. N. Y.) 1926, A.M. (Columbia) 1930. Dir. of Speech Clin. and Instr. in Speech Cor., Main Center, City Coll., 139th St. and Convent Ave., New York City.

(A) FINGER, VERNA, S.B. (Northwestern Univ.) 1931, S.M. (ibid) 1932. Instr. of Speech Re-educ., Northwestern Univ. 31 E. Division, Fond du Lac, Wis.

(A) FISHEL, MAMIE VAUGHN, S.B. (Meridian) 1913, Ph.M. (Wis.) 1935. Pub. Sch. Sup. of Speech Cor. 16 Marshall Pl., Janesville, Wis.

(A) FISK, JEANNE BISHOP (Mrs. Robert S. Fisk), A.B. (Rockford) 1935, A.M. (Columbia) 1936. Pub. Sch. T. of Speech Cor., Minneapolis, Minn. 3959 Quail Ave., Robbinsdale, Minn.

(F) FITZ-GIBBON, JOHN J., F.A.C.D. (Am. Coll. of Dentists) 1928. Chief of Dental Staff, Skinner Memorial Clin., Holyoke Hosp.; Consultant and Palate Prosthetist, Joseph Samuels Dental Clin., R. I. Hosp. Private Practice, 56 Suffolk St., Holyoke, Mass.

(A) FRASIER, JEANNETTE, M.A. (Univ. of Iowa) 1940. T. Speech Cor., Davenport Pub. Schools. 819 Broadway, Davenport, Iowa.

(F) FROESCHELS, EMIL, M.D. (Vienna Univ.) 1907. 133 East 58th St., New York, N. Y.

(A) GAINES, FRANCES PERLOWSKI (Mrs.), A.B. (Wis.) 1926, S.S.M. (Smith) 1927. Dir. of Speech Clin., Children's Memorial Hosp., Chicago, and Rush Medical Coll., Chicago. 548 Fullerton Pkwy., Chicago, Ill.

(A) GALLANT, MARJORI L., B.A. (Hunter Coll.) 1937, M.A. (Iowa) 1939. Asst. Speech Clin., Purdue Univ. 121 Wiggins St., West Lafayette, Ind.

(F) GARDNER, WARREN HENRY, A.B. (Harvard) 1918, Ph.D. (Iowa) 1936. Hearing Examiner, Speech and Hearing Clin., Indiana Univ., Bloomington, Ind.

(A) GARRISON, E. GERALDINE, A.B. (Okla. State) 1925, A.M. (Columbia) 1938. Dir. of Speech Activities. 917 N. 8th St., Neodesha, Kansas.

(A) GEESON, EDNA LABUHN (Mrs. Edward Geeson), S.B. (Wayne) 1928, A.M. (ibid) 1933. T. of Speech Imp., Cass High Sch. 420 Navahoe Ave., Detroit, Mich.

(F) GIFFORD, MABEL FARRINGTON (Mrs.). Chief, Bureau of Cor. of Speech Defects, Calif. State Dept. of Educ.; Dir. of Speech Clin. and Instr. in Speech Cor., Univ. of Calif. and San Francisco State T. Coll. 609 Sutter St., San Francisco, Calif.

(A) GILLETT, ANNA MAE, A.B. (Univ. Wichita) 1938. Sup. in Speech Clin. 1751 N. Waco, Wichita, Kansas.

(A) GILMAN, WILBUR ELWYN, A.B. (Cornell) 1923, Ph.D. (ibid) 1937. Assoc. Prof. of Speech, Univ. of Mo. 512 Steward Road, Columbia, Mo.

(A) GLASNER, PHILIP J., B.C.S. (Baltimore Coll. of Commerce) 1932. Speech Correctionist, Johns Hopkins Hosp., Psychiatric Clin.; Dir. of Private Clin., 740 Reservoir St., Baltimore, Md.

(A) GLASPEY, ESTHER, A.B. (Iowa) 1932, A.M. (ibid) 1933. Pub. Sch. T. of Speech Cor., Lecturer in Extension at Butler Univ. 150 N. Meridian, Indianapolis, Ind.

(A) GLAUBER, I. PETER, M.D. (Univ. and Bellevue Hosp. Med. Coll.) 1925. 111 East 60th St., New York City.

(F) GOLDSTEIN, MAX A., M.D., F.A.C.S., LL.D. (Washington Univ.) 1892. Dir., Central Inst. for the Deaf. 4574 W. Papin St., St. Louis, Mo.

(A) GOODFRIEND, DAVID JOSEPH, D.D.S. (Univ. of Penn.) 1919. Researcher, Univ. of Pennsylvania. 901 Medical Art Bldg., Philadelphia, Pa.

(A) GOODWIN, STELLA ULRICH (Mrs. Clyde Goodwin), A.B. (Wayne Univ.) 1936. Pub. Sch. T. of Speech Cor. 4404 Somerset, Detroit, Mich.

(A) GORDON, EVELYNE FRANCES (Mrs. Frederick C. Gordon), S.B. (Columbia) 1936, A.M. (ibid) 1937. Director of Speech Clinic, Long Beach High School, Long Beach, L. I., New York.

(A) GOTTLOBER, ABRAHAM BER., A.B. (Adelbert Coll., Western Reserve Univ.) 1933, A.M. (Graduate Coll., W. R. U.) 1935, Ph.D. (Iowa) 1937. Dir. Cleveland Sp. Cor. Clinic, Visitant to City Hospital, 3608 Euclid Ave., Cleveland, Ohio.

(A) GRAY, MARCELLA ELIZABETH, A.B. (Iowa) 1937, M.A. (ibid) 1940. Supervisor, Corrective Speech, West Waterloo Schools. 119 East Davenport St., Iowa City, Iowa.

(F) GREENE, GUY SHEPARD, A.B. (Hobart) 1920, Ph.D. (Cornell) 1926. Dir. of Speech Clin. and Prof. of English and Head of the Dept. of Eng. and Speech, Iowa State Coll., Ames, Iowa.

(A) GREENOUGH, JEANNETTE B., A.B. (Denison Univ) 1931, A.M. (T. Coll., Columbia) 1934. T. of Speech at Private Clin. 49 Prospect St., E. Orange, N. J.

(A) GRIFFITH, FRANCIS, A.B. (St. John's Univ., Brooklyn) 1927, A.M. (ibid) 1929. Chairman, Eng. Dept., James Madison High Sch. 1751 E. 29th St., Brooklyn, N. Y.

(A) GROSS, HILDRED AUGUSTA, A.B. (Mich.) 1931, A.M. (ibid) 1934. Pub. Sch. T. of Speech Cor. 899 Virginia Pk., Detroit, Mich.

(A) GUDRIDGE, HAZEL LUNDSTEN (Mrs. F. M. Gudridge), A.B. (Macalester) 1922. Pub. Sch. T. of Speech Cor. 189 Amherst Str., St. Paul, Minn.

(A) HAHN, EUGENE F., A.B. (Univ. Calif.) 1929, A.M. (Univ. S. Calif.) 1933, Ph.D. (ibid) 1940. First Asst. Supervisor of Language Educ., Detroit Pub. Schs.; Asst. Prof. of Speech Educ., Dir. of Speech Clin., Wayne Univ., Detroit, Mich.

(F) HALL, MARGARET ELIZABETH, A.B. (Wis.) 1927, S.M. (Univ. Chicago) 1931, Ph.D. (Iowa) 1938. Psychologist and Speech Pathologist, Bureau of Child Study, Chicago Board of Educ. 228 N. La Salle St., Chicago, Ill.

(A) HALSTED, ELIZABETH MARGARET. Pub. Sch. T. of Speech Cor. 2755 Franklin St., San Francisco, Calif.

(A) HAMBLIN, MARION JUNE, B.L.I. (Emerson) 1935. Pub. Sch. T. Speech Cor. 10 Clisbee Sq., Revere, Mass.

(A) HAMILTON, RUTH GREENWOOD, Ed.B. (R. I. Coll. Ed.) 1931. Pub. Sch. T. Hope Valley, R. I.

(A) HANNING, LUELLA MARIE. T. in Duluth Pub. Sch., Adm. Bldg., Duluth, Minn.

(A) HANSEN, BURRELL F., B.S. (Utah State Agr. Coll.) 1940. Asst. Prof. of Speech, Purdue Univ. 209 E. Lutz Ave., West Lafayette, Indiana.

(A) HARKINS, CLOYD S., A.B. (Univ. of Pittsburgh), D.D.S. (ibid). Consultant to Penn. State College Speech Clinic. Fulton Bldg., Osceola Mills, Penn.

(F) HAWK, SARA STINCHFIELD (Mrs. Charles Lyle Hawk), A.B. (Pittsburgh) 1914, A.M. (Iowa) 1920, Ph.D. (Wis.) 1922. Psychologist, Pasadena Polytech. Elem and Jr. H. S.; Dir. of Speech Clin., Orthopedic Hosp. and Child Guidance Clin. of Los Angeles and Pasadena; Lect. on Psychol. and Speech Pathol., Univ. of S. Calif. 1577 N. Gower St., Hollywood, Calif.

(A) HEDGES, THADINE, A.B. (Wichita Univ.) 1939. Supervisor in Speech Clin., Institute of Logopedics. 1811 N. Hillside, Wichita, Kansas.

(A) HEDRICK, JENNIE. Prin. Washington Sch. for the Cor. of Speech Defects. 3321 N. St., Washington, D. C.

(F) HELTMAN, HARRY JOSEPH, S.B. (Syracuse) 1910, A.M. (American) 1920. Chairman, Sch. of Speech & Dramatic Art and Prof. of Speech, Syracuse Univ., Syracuse, N. Y.

(A) HENDERSON, ELLEN CLARK (Mrs.), A.B. (Utah) 1911, A.M. (T. Coll., Columbia) 1925. T., Chicago School of Drama and Music. Editor of Speech Magazine. 537 South Dearborn (1102) Chicago, Ill.

(F) HENRIKSON, ERNEST H., A.B. (Ore.) 1925, A.M. (Iowa) 1929, Ph.D. (ibid) 1932. Dir. of Speech Cor. and Assoc. Prof. of Speech, Iowa State T. Coll., Cedar Falls, Iowa.

(A) HERENDEEN, HARRIET, S.B. (Fremont Nor., Nebr.) 1906, A.B. (Wyo.) 1929. Special Educ. 2094 Neil Ave., Columbus, Ohio.

(A) HERMAN, IRENE M., B.S. (Fordham) 1932. T., Speech Cor., Yonkers Pub. Schools. 75 Bruce Ave., Yonkers, N. Y.

(A) HERTLEIN, LOIS ELLEN, B.S. (College of Emporia) 1938. Instr., Institute of Logopedics. 1751 Fairmount, Wichita, Kansas.

(A) HIAR, IRMA THERESSA, S.B. (Mich. State Normal) 1934. T., Speech Cor., Lansing Pub. Schools. 314 Townsend, Lansing, Mich.

(A) HIBBITT, GEORGE WHITING, A.B. and S.B. in Ed. (Ohio State) 1920, A.M. (ibid) 1922. Assoc. in English, Columbia Univ. 456 Riverside Drive, New York City.

(A) HIGGINS, ROGER WOLCOTT, A.B. (Clark Univ.) 1923, A.M. (Harvard) 1931. Dir. of Speech Clin. and Instr. in English and Pub. Speaking, Phillips Academy, Andover, Mass.

(A) HISER, VELMA BISSELL, L.B. (Northwestern) 1926, A.M. (Iowa) 1938. Dir. of Speech Clin., Grinnell College. Hotel Monroe, Grinnell, Iowa.

(A) HOLWAY, DORA EDYTHE (Mrs.), S.B. (Wayne Univ.) 1931. T. of Speech Cor. at Detroit Public Schools. 14015 Rutland, Detroit, Mich.

(A) HOMONOFF, BEATRICE, A.B. (Emerson) 1938. Instr. in Speech Cor. in Brockton, Mass., Pub. Sch.; Speech Pathologist of the Brockton Habit Clinic. 97 Hazelton St., Mattapan, Mass.

(A) HOUSE, ENOD SCOVILLE (Mrs.), A.B. (Iowa) 1930, A.M. (ibid) 1936. Pub. Sch. T. of Speech Cor. 5515 Sheridan Rd., Kenosha, Wis.

(A) HOWARD, KATHRYN LOUISA, A.B. (Mt. Holyoke) 1936. T. of English, Speech Cor., and Dramatics, Pierce Coll., Elleniko, Glyfada, Greece.

(A) HOWE, AGNES SMART (Mrs.), B.L.I. (Emerson) 1925. Dir. of Psychological Testing and Speech Correction, Children's Service Bureau; Speech T., State Dept. of Educ.; Pathologist, Bay Pines Hospital; Dir., The School of Speech, 302 Central Ave., St. Petersburg, Florida.

(A) HULL, HENRIETTA C., Sc. B. (Minn.) 1936. Pub. Sch. T. of Speech Cor.; Grad. Asst. in Speech Pathol., Univ. of Minn., Minneapolis. 323 W. Broadway, Winona, Minn.

(A) HULTZEN, LEE S., A.B. (Cornell) 1920, Ph.D. (ibid) 1932. Dir. of Speech Clin. and Assoc. Prof. of Speech, Univ. of Missouri. 309 Switzler Hall, Columbia, Missouri.

(A) HUNT, JAMES FRANKLIN, S.B. (Univ. Penna.) 1931, S.M. (Mass. State Coll.) 1935. Dir. of Speech Clin. of Holyoke, Mass., Child Guidance Clin.; Instr. in Spoken English at Smith Coll. and of Pub. Speaking at Amherst Coll. 21 Taylor St., Amherst, Mass.

(A) HUNZIKER, MINNA ELIZABETH, A.B. (Western State T. Coll.) 1929, A.M. (Northwestern) 1940. Speech Cor. T., Pub. High Schools. 49 Union Ave., N. E., Grand Rapids, Mich.

(A) HURD, MELBA FRANCES, A.B. (Minn.) 1927, A.M. (ibid) 1931. Instr. in Speech, Univ. of Minn. 819 Essex St., S. E., Minneapolis, Minn.

(A) HUYCK, E. MARY, A.B. (Univ. S. Dak.) 1934, A.M. (Univ. Denver) 1937. Instr. in Speech, Univ. of Ariz. 1215 N. Euclid, Tucson, Ariz.

(A) HYDE, RUTH STUTENROTH (Mrs.), A.B. (Univ. S. Dak.) 1923, A.M. (Iowa) 1937. 127 Iroquois St., Laurium, Mich. (Calumet).

(A) INNESS, MARJORY A., A.B. (Iowa) 1937, A.M. (ibid) 1938. Speech Correctionist, East Chicago Public Schools. 4819 Magoun Ave., East Chicago, Indiana.

(A) ISRAEL, EVA, A.B. (Brown Univ.) 1929. T., Speech Cor., Dramatics and Public Speaking in the Senior High School, Woonsocket, R. I. 563 S. Main St., Woonsocket, Rhode Island.

(A) JACOB, MARY JANE, B.S. (Wichita Univ.) 1939. Instr., Cor. Speech, Institute of Logopedics. 220 Parkwood Lane, Wichita, Kansas.

(A) JACOBY, BEATRICE FRANCIS, A.B. (Hunter Coll.) 1933, A.M. (Columbia Univ.) 1934. Instr. in Speech, Queens Coll. 7 W. 96th St., New York City.

(A) JELLINEK, AUGUSTA, Ph.D. (Univ. of Vienna) 1923. Asst. Dir. of Speech Clinic, Prof. of Speech Therapy, Washington Univ. 818 South Kingshighway, St. Louis, Mo.

(A) JEROME, ELDON K., S.B. (Northwestern) 1932, A.M. (ibid) 1936. Asst. Dir. of Speech Clin., Purdue Univ., Lafayette, Ind.

(F) JOHNSON, WENDELL A. L., A.B. (Iowa) 1928, A.M. (ibid) 1929, Ph.D. (ibid) 1931. Assoc. Prof. of Psychology and Speech Pathol., East Hall, Univ. of Iowa, Iowa City, Iowa.

(A) JONES, AMY DESMOND, A.B. (Univ. Wichita) 1937. Instr. in Speech Cor. at The Cedars, Ross, Calif.

(A) JONES, MAMIE JOSEPHINE, A.B. (Huntington) 1936, A.M. (Northwestern) 1937. Georgia State T. Coll., Statesboro, Ga.

(A) JONES, MARTHA ELIZABETH, Ed.B. (T. Coll. of Conn.) 1937. Pub. Sch. T. of Speech Cor. 282 C Sigourney St., Hartford, Conn.

(A) JOSLIN, NORA M., S.B. (Western Reserve) 1934, A.M. (Columbia) 1938. Pub. Sch. T. of Speech Cor. and Lip Reading, Cleveland, Ohio. 3155 Rocky River Drive, Cleveland, Ohio.

(A) KANE, LILLIAN, S.B. (N. Y. Univ.) 1929, A.M. (Columbia Univ.) 1932. Dir. of Speech Clin. and Pub. Sch. T. of Speech Cor., Montclair N. J. 679 E. 23rd St., Paterson, N. J.

(F) KANTNER, CLAUDE E., A.B. (Albion) 1928, A.M. (Wis.) 1930, Ph.D. (ibid) 1933. Dir. of Speech Clin. and Assoc. Prof. of Speech, La. State Univ., University, La.

(A) KEASTER, JACQUELINE ALCINDA. T. of Speech Cor., Central Institute for the Deaf, 818 S. Kingshighway, St. Louis, Mo.

(A) KEELER, DARLENE DUKE (Mrs. Irvin F. Keeler), A.B. (Iowa) 1930, A.M. (ibid) 1935. 133 S. Loomis St., Naperville, Ill.

(A) KELLENBERGER, JEANNE, A.B. (Iowa) 1933, A.M. (ibid) 1937. 730 E. High St., Davenport, Iowa.

(F) KELLY, GEORGE A., A.B. (Park Coll.) 1926, A.M. (Kansas) 1928, B.Ed. (Edinburgh) 1930, Ph.D. (Iowa) 1931. Dir., Psychol. Clin., Ft. Hays Kansas State Coll. 710 Park Ave., Hays, Kansas.

(F) KENNEDY, LOU, A.B. (Iowa) 1911, A.M. (Stanford) 1922, Ph.D. (Wis.) 1930. Dir. of Speech Clin. and Assoc. Prof. of Speech, Brooklyn Coll., Bedford Ave. and Ave. H, Brooklyn, N. Y.

(A) KENNEDY, MARION ROSALIE, A.B. (Our Lady of the Elms Coll.) 1937. T. of Speech Cor., Holyoke Public Schools. 30 Myrtle Ave., Holyoke, Mass.

(F) KENYON, ELMER LAWTON, A.B. (Harvard) 1890, M.D. (Rush) 1896. Assoc. Prof. of Otolaryngology, Rush Medical Coll., Univ. of Chicago, Clinical Emeritus. Private Practice, 185 North Wabash Ave., Chicago, Ill.

(A) KIMMELL, MELBA LOUISE, S.B. (Northwestern) 1936, A.M. (Iowa) 1937. 18 Hampton Rd., Eastborough, Wichita, Kansas.

(A) KIRBY, A. FLORENCE, B.S. (Clark Univ.) 1935, M.A. (Boston Univ.) 1937. Prof. Speech and Literature, State Teachers College, Lowell, Mass. 52 Livingston Ave., Lowell, Mass.

(A) KIRK, KATHRYN. Speech Examiner and Demonstration Tr. of Speech Cor. in Elem. Sch. 181 Kirkland Rd., Rochester, N. Y.

(A) KLINGBEIL, HENRY CLAYTON, A.B. (Penn. State) 1916, A.M. (Mich.) 1927. Instr. in Speech, Jr. Coll. 2136 Sixth St., Bay City, Mich.

(A) KLUEVER, LOIS H. COBB (Mrs. H. C. Kluever), A.B. (Iowa) 1927, A.M. (ibid) 1931. Clin. Asst. in Speech and Otolaryngology, Univ. of Iowa, Iowa City, Iowa. 731 Warden Apts., Ft. Dodge, Iowa.

(A) KNIGHT, BILLIE BRANDT, S.B. (Northwestern) 1937. Private practice at 210 Medical Arts Bldg., Houston, Texas.

(A) KNIGHT, HELEN SULLIVAN (Mrs. Paul D. Knight), A.B. (Arkansas Coll.) 1931, A.M. (Northwestern) 1936. Instr. in Speech Re-educ., Northwestern Univ., Evanston, Ill.

(A) KNIGHT, PAUL D., S.B. (Ill.) 1926, A.M. (Northwestern) 1940. Private practice in speech disorders, 55 E. Washington St., Chicago, Ill.

(A) KNUDSON, THELMA A., S.B. (Northwestern) 1928, A.M. (Iowa) 1937. Dir. Speech Clinic and Instr. in Speech, John Adams H. S., S. Bend, Indiana.

(F) KOEPP-BAKER, HERBERT, A.B. (Mich.) 1926, S.M. (Penn. State) 1930, Ph.D. (Iowa) 1938. Dir. of Speech Clin., Pa. State Coll., State College, Pa.

(F) KOPP, GEORGE ADAMS, S.B. (Monmouth) 1926, S.M. (Wis.) 1930, Ph.D. (ibid) 1933. Assoc. Prof. of Speech Educ. and Dir. of Speech Laboratories, Teachers Coll., Columbia Univ. 156 Ames Ave., Leonia, N. J.

(A) KOSH, ZELDA HORNER (Mrs. David A. Kosh), A.B. (Adelphi Coll.) 1933, A.M. (T. C., Columbia) 1934. T. of Speech at Girls High Sch. 55 Pineapple St., Brooklyn, N. Y.

(A) KRAFT, MARIE BERGIN (Mrs.), S.B. (Ithaca Coll.) 1932, A.M. (Columbia Univ.) 1935. Instr. of Speech Cor. and Speech Educ., Extension Div., Brown Univ. 97 Hilltop Ave., Providence, R. I.

(A) KRAMER, MAGDALENE, A.B. (Trinity Coll., Wash. D. C.) 1920, A.M. (Columbia) 1930, Ph.D. (ibid) 1936. Assoc. Prof. of Speech, T. Coll., Columbia Univ., New York City.

(A) LASSE, LEROY, Ph.D. (Univ. of Iowa) 1937. Head Dept. of Speech and Dir. Speech Clin., Univ. of Nebraska, Lincoln, Nebr.

(A) LETZTER, MARGARET CATHERINE, A.B. (Northwestern Univ.) 1915, A.M. (ibid) 1930, Ph.D. (ibid) 1937. Dir. of Speech Clin. and Asst. Prof. of Speech, San Jose State Coll. 55 S. 6th St., San Jose, Calif.

(A) LEVBARG, JOHN JACOB, M.D. (L. I. Med. Coll.) 1913. Dir. Speech Clin., Harlem Eye & Ear Hosp.; Instr. in Voice Science and Speech Pathol., New York Bd. of Educ., Diplomate of Otolaryngology. 55 W. 42nd St., New York City.

(A) LEVY, ELEANOR SELMA, B.A. (Emerson Col.) 1940. Dir. of Speech Clinic, Mass. Division of Mental Hygiene. 179 Rawson Road, Brookline, Mass.

(A) LEVY, RAYE CHARLOTTE. Pub. Sch. T. of Speech Cor. 5507 Stanton Ave., Pittsburgh, Pa.

(A) LEWIS, FLORENCE STILES (Mrs.) B.L.I. (Emerson) 1937. Speech Cor. T., State Dept. Health, Hartford Connecticut.

(A) LILJEBERG, NAOMI, S.B. (Minn.) 1939. Pub. Sch. T. of Speech Cor. 1924 E. River Terrace, Minneapolis, Minn.

(A) LINKOW, IRVING, A.B. (Univ. Denver) 1937, A.M. (ibid) 1939. Instr. in Speech, Dept. of English and Speech, Iowa State Coll., Ames, Iowa.

(A) LINSCOTT, ANNE M., B.L.I. (Emerson) 1940. Private Speech Cor. Studio, 78 Greenwood Ave., Swampscott, Mass.

(A) LOMBARD, ELSIE ANDERSON (Mrs.), A.B. (Macalester) 1923. Pub. Sch. T. of Speech Cor. 1490 E. 7th St., St. Paul, Minn.

(A) LUNDSTEN, HAZEL VIVIAN (Mrs. F. M. Gudridge).

(A) MCCALMON, GEORGE ALEXANDER, A.B. (Carnegie Insti. of Technology) 1934, A.M. (Columbia) 1938. Prof. of Speech, Geneva College, Beaver Falls, Pa.

(A) MCCAUSLAND, MARGARET, B.S. (Univ. of Penna.) 1939. T., Speech Improvement in ten Philadelphia Public Schools. 6139 Christian St., Philadelphia, Pa.

(A) MCCLELLAND, VERA, Ph.B. (Univ. Chicago) 1923. Kindergarten T. Hotel Carter, Cleveland, Ohio.

(A) MCCONECHY, KATHARINE MARIA. Instr. in Dept. of Speech and Expression, Regina Coll., Univ. of Sask. 3031 Rae St., Regina, Sask., Canada.

(A) MCCORD, EDNA MORRIS (Mrs.), A.B. (Hunter) 1903. Pub. Sch. T. of Speech Imp. 825 W. 187th St., New York City.

(A) MACDOUGALL, ANNE DORIS, B.L.I. (Emerson) 1936. T. of Pub. Speaking in High Sch. 62 Maplewood Ave., Hempstead, N. Y.

(F) MCDOWELL, ELIZABETH DICKINSON (Mrs. Sam B. McDowell), A.B., S.B. (Judson) 1914, A.M. (Columbia) 1920, Ph.D. (ibid) 1928. Lahaska, Bucks County, Pa.

(F) MCGINNIS, MILDRED A., S.B. (Washington Univ.) 1933, A.M. (ibid) 1939. Sup. T. of Speech Development and Cor., Univ. Coll., Washington Univ.; Dir. of Speech Clin., Central Inst. for the Deaf. 818 S. Kingshighway, St. Louis, Mo.

(A) MACLEARIE, ELIZABETH C., A.B. (Ball State T. Coll.) 1925, A.M. (Wis.) 1935. Pub. Sch. T. of Speech Cor., Cleveland Heights, Ohio. 2366 Noble Road.

(A) MCMURRY, OCEA, A.B. (Coll. of Pacific) 1926. Co. Sup. of Speech Cor. and Speech Imp., Shasta Co., Calif. Box 956, Redding, Calif.

(A) MADDOX, JAMES S., A.B. (W. Va. Univ.) 1935, A.M. (Iowa) 1937. Speech Clin. Staff and Instr. in Speech, Purdue Univ., Lafayette, Ind.

(A) MAJOR, CHARLES C., A.B. (Carthage Coll.) 1933, M.S. (Purdue) 1940. Dir. of Speech Clin. and Instr. Speech and English, Ill. Wesleyan Univ. 512 N. Roosevelt, Bloomington, Illinois.

(A) MANCHESTER, MARY JANE, A.B. (Kent State Univ.) 1933, M.S. (Mich.) 1934. T. of Corrective Speech, Highland Park, Mich.; 11 McLean Ave., Highland Park, Mich.

(A) MANIFOLD, AGNES D. (Mrs.), A.B. (St. Joseph's Coll. for Women) 1926. T. of Speech, Flushing High Sch. 35-17 146th St., Flushing, N. Y.

(A) MARCERO, FRANCIS ALOYSIUS, A.B. (Mich.) 1938. T. of Speech Cor. Placement, Detroit. 1445 Bishop Rd., Grosse Pointe, Mich.

(A) MARTIN, SUZANNE ELIZABETH, A.M., Ed.D. (Columbia) 1933, 1939. (Bender, Blanton.) N. Y. Univ., Brooklyn Coll. 110-21 73rd Rd., Forest Hills, Long Island, N. Y.

(A) MASE, DARREL JAY, S.B. (Emporia State T. Coll., Kans.) 1928, A.M. (Mich.) 1932. Dir. Speech Clin., Calif. State T. Coll., Pa. 813 Fifth St., California, Pa.

(A) MASLAND, MARY WOOTTON (Mrs.), S.B. (McGill Univ.) 1935, A.M. (Oberlin Coll.) 1939. Private Practice, 222 So. 39th St., Philadelphia, Pa.

(F) MASON, MARIE KATHERINE, A.B. (Canisius) 1924, A.M. (Ohio State) 1932. Asst. Dir. of Speech Clin. and Asst. Prof. of Speech and Visual Hearing, Ohio State Univ. 60 E. Norwich Ave., Columbus, Ohio.

(A) MAYER, M. GLADYS, S.B. (Wayne) 1932, S.M. (Univ. Detroit) 1936. Pub. Sch. T. of Speech Cor. 16847 Baylis, Detroit, Mich.

(F) MEYER, MAX FREDERICK, Ph.D. (Berlin), 1896. Research Prof. of Psychol., Univ. of Miami. 3939 Loquat, Coconut Grove, Fla.

(A) MIANO, KATE PUPELLA (Mrs.). Pub. Sch. T. 517 80th St., Brooklyn N. Y.

(A) MICHLINSKI, FLORENCE EVA, A.B. (Mich.) 1939, M.A. (ibid) 1940. T., Speech Cor., Louisville Pub. Schools. 124 W. Ormsby, Louisville, Ky.

(F) MILISEN, ROBERT LLOYD, A.B. (Simpson) 1931, A.M. (Iowa) 1933, Ph.D. (Iowa) 1937. Indiana Univ., Bloomington, Ind.

(A) MILLER, ELVENA, A.B. (Wash.) 1917, A.M. (ibid) 1936. Pub. Sch. Sup. of Speech. 7330 Fifteenth Ave., N. E., Seattle, Wash.

(A) MILLS, ALICE W. (Mrs.), S.B. (Ore.) 1905, A.M. (Iowa) 1923. Dir. of Speech, Assoc. Prof., Mt. Holyoke Coll., South Hadley, Mass.

(A) MILWARD, ELEANOR JANE, S.B. (Northwestern Univ.) 1936. Dir. of Speech Clin. at James Whitcomb Riley Hosp.; Speech Therapist, Cerebral Palsy Project, Ind. State Dept. of Pub. Welfare. 1401 N. Pa., Indianapolis, Ind.

(A) MOORE, ROBERTA, A.B. (Fla. State Coll.) 1932, A.M. (Iowa) 1939. Pub. Sch. T. of Speech Cor., S. Beach Sch., Miami Beach, Florida.

(F) MOORE, WILBUR ERWIN, A.B. (Mo. Wesleyan) 1926, A.M. (Iowa) 1932, Ph.D. (ibid) 1936. Head Dept. of Speech, Central State Teachers, Mich. 812 S. College, Mt. Pleasant, Mich.

(A) MOREN, J. ADELAIDE, S.B. (Minn.) 1938. Asst. Dir. of Speech Clin., Univ. Hosp., 1354 Arcade St., St. Paul, Minn.

(F) MORLEY, ALONZO JOHN, A.B. (Brigham Young) 1925, A.M. (ibid) 1931, Ph.D. (Iowa) 1935. Assoc. Prof. of Speech, 180 College Bldg., Brigham Young Univ., Provo, Utah.

(F) MORRIS, D. W., A.B. (Park Coll.) 1928, A.M. (Maine) 1934, Ph.D. (Iowa) 1936. Dir. of Speech and Reading Clin. and Chair. Dept. of Speech, Ind. State T. Coll. 2508 Farrington, Terre Haute, Ind.

(A) MORRIS, JOHN ANDREW, B.S. (Phillips Univ.) 1929, M.A. (Iowa) 1939, M.A. (Phillips) 1930, B.D. (Yale) 1932. Asst. Prof. of Speech, Phillips Univ. 2110 East Broadway, Enid, Oklahoma.

(F) MOSER, HENRY MICHAEL, A.B. (Ohio State) 1924, A.M. (Mich.) 1927, Ph.D. (Iowa) 1936. Asst. Prof. of Speech Cor., Univ. of Mich. 1024 Baldwin Ave., Ann Arbor, Mich.

(A) MOSES, ELBERT RAYMOND, Jr., A.B. (Univ. Pittsburgh) 1932, S.M. (Mich.) 1934, Ph.D. (ibid) 1936. Asst. Prof. of Speech, Ohio State Univ. 4499 Rosemont Pl., Columbus, Ohio.

(A) MOUSER, WILLIAM S., A.B. (Wayne Univ.) 1935, A.M. (ibid) 1939. Dir. of Speech Clin., Grace Hosp., Detroit. 14845 Ashton Rd., Detroit, Mich.

(A) MUELLER, HAZEL DU CLES (Mrs. F. W. Mueller, Jr.), S.B. (Northwestern) 1930, S.M. (ibid) 1935. Speech Counselor, Kenilworth and Wilmette Schools. 2255 Ridge Ave., Evanston, Ill.

(F) MURRAY, ELWOOD, A.B. (Hastings) 1922, A.M. (Iowa) 1924, Ph.D. (ibid) 1931. Chair. Dept. of Speech and Dramatic Arts, Univ. of Denver. 2391 S. Clayton St., Denver, Colo.

(A) NAGY, HELLEN MARIE, A.B. (Hunter Col.) 1937. Dir. of Speech Clin. and Prof. of Remedial Speech, N. Y. Institute for the Blind. 999 Pelham Parkway, New York, N. Y.

(F) NATHANSON, YALE, Sc.B. (Univ. Penn.) 1923, A.M. (ibid) 1924, Ph.D. (ibid) 1930. Dir. of Speech Clin., St. Christopher's Hosp. for Children; Chief Psychol., Philadelphia Psychiatric Hosp. Private Practice, Central Medical Bldg., 18th and Chestnut Sts., Philadelphia, Pa.

(A) NEELY, BETTY JANE, B.E. (Wichita Univ.) 1938. Instr. in Speech Cor., Wichita Univ. 1327 Lane, Topeka, Kansas.

(A) NELSON, SEVERINA ELAINE, A.B. (Univ. Ill.) 1918, A.M. (ibid) 1923, Ph.D. (Wis.) 1938. Dir. of Speech Clin. and Assoc. in Speech, Univ. of Ill. 715 Vermont St., Urbana, Ill.

(F) NEMOY, ELIZABETH MCGINLEY (Mrs.), A.B. (Temple) 1928, A.M. (Penn.) 1931. Pub. Sch. T. of Speech Cor. The Fairfax, 43rd and Locust, Philadelphia, Pa.

(A) NEVINS, MARXINE BERNICE, A.B. (Washburn Col.) 1933. Instr. in Speech Cor., Univ. of Wichita. 1005 Central, Dodge City, Kansas.

(F) NEWHART, HORACE, A.B. (Dartmouth) 1895, M.D. (Mich.) 1898. Prof. of Otolaryngology, Univ. of Minn. 527 Medical Arts Bldg., Minneapolis, Minn.

(A) NORCROSS, MABEL CLARE (Mrs. Edward P. Norcross), Ph.B. (Univ. Chicago) 1928, A.M. (Columbia Univ.) 1930. T. of Speech Cor., Elem. Sch. Private Practice, 515 Maple Lane, Highland Park, Ill.

(A) NORRIS, MARION A., A.B. (Iowa) 1933, A.M. (ibid) 1934. Pub. Sch. T. of Speech Cor., 1009 Lincoln, Superior, Wis.

(A) NYSTROM, CLARENCE L., A.B. (Greenville) 1925, A.M. (Iowa) 1929, Ph.D. (ibid) 1932. Dir. of Speech Clin., and Assoc. Prof. of Speech, Wheaton Coll. 708 Irving Ave., Wheaton, Ill.

(A) OBERMANN, C. ESCO, A.B. (Iowa) 1926, A.M. (ibid) 1932, Ph.D. (ibid) 1938. Research Fellow in Child Psychol., Graduate Coll., Univ. of Iowa, Iowa City, Iowa.

(A) OGILVIE, MARDEL, A.B. (Cornell Univ.) 1931, A.M. (ibid) 1932. T. of Speech, Fredonia State Normal Sch., Fredonia, N. Y.

(A) OLIVER, DOROTHY, S.B. (Northwestern) 1935. 744 Van Buren, Gary, Indiana.

(A) OSBORN, COURTNEY DALE, A.B. (Univ. Wichita) 1936, A.M. (ibid) 1939. Instr. in Speech Clin., Univ. of Wichita. 1728 N. Fairmount, Wichita, Kansas.

(A) OSGOOD, RUDOLF, S.B. (Harvard) 1924, M.S. (Rush Med. School) 1932. Dir. of Speech Clinic, Phillips Academy, Andover, Mass. 370 Common Street, Belmont, Mass.

(A) PALKOT, EDWARD JOHN, B.S. (Carnegie Tech.) 1935, M.A. (Columbia) 1940. Dir. of Speech Clin., and T. of Speech, Garden City Schools. 73 Garden St., Garden City, New York.

(F) PALMER, MARTIN FRANKLIN, A.B. (Olivet) 1927, A.M. (Mich.) 1931, Sc.D. (ibid) 1937. Dir. Institute of Logopedics; Prof. and Chairman, Dept. of Logopedics, Univ. of Wichita. 522 S. Belmont, Wichita, Kansas.

(A) PAMKASKIE, MARGARET, A.B. (Ft. Hays, Kansas, Stat Coll.) 1937, M.A. (ibid) 1938, Ph.D. (Iowa) 1941. Instr. in Speech and Asst. Dir. Speech and Reading Clin., Ind. State T. Coll. 1908 So. 7th St., Terre Haute, Indiana.

(F) PARDOE, T. EARL, A.B. (Brigham Young) 1924, A.M. (S. Calif.) 1931, Ph.D. (La. State) 1936. Prof. of Speech, Brigham Young Univ. 160 S. 1st E. St., Provo, Utah.

(A) PATTERSON, JEAN, B.L.I. (Emerson) 1940. Sup. of Speech Cor. in Child Guidance Speech Clinics, Boston. 33 S. Maple St., Westfield, Mass.

(A) PAULS, MIRIAM DOROTHY, A.B. (Harris T. Coll.) 1932, A.M. (Wayne Univ.) 1939. T. in Speech and Lip Reading, N. J. School for Deaf. 4972 Itaska Ave., St. Louis, Mo.

(A) PETERSON, GORDON E., A.B. (DePauw Univ.) 1935, M.A. (Louisiana State Univ.) 1937, Ph.D. (ibid) 1939. Dir. of Speech Clin. and Prof. of Speech, Ball State T. Coll. 1408 West Main St., Muncie, Ind.

(A) PFAFF, PAUL LEWIS, A.B. (Stanford) 1929, A.M. (Univ. S. Calif.) 1935. Dir. of Speech Clin. and Asst. Prof. of Speech, San Diego State Coll., San Diego, Calif.

(A) PIERCE, L. ADELINE, A.B. (Albion Coll.) 1930, M.A. (Columbia) 1931, Ph.D. (Mich.) 1940. Dir. of Speech Clin. and Dir. of Speech Activities, Lewis Institute—Speech Center. 59 Massachusetts, Detroit, Michigan.

(A) PITTELMAN, RUTH KAY, S.B. (Wis.) 1939. Asst. Dir. of Speech Clin., Purdue University, West Lafayette, Indiana.

(A) PITTENGER, KATHERINE LYDIA, S.B. in Ed. (Texas) 1937, A.M. in Speech (Iowa) 1939. Private Practice of Speech Cor. 822 E. 37th St., Austin, Texas.

(A) PLUMLEY, HARRIET ELIZABETH, B.O.E. (Syracuse) 1927. T. of Speech Cor., Niagara Falls Public Schools. 416 Pine Ave., Niagara Falls, New York.

(A) POWERS, DAVID GUY, A.M., Ed.D. (Columbia) 1934, 1938. Instr. in Speech, Queens Coll. 110-21 73rd Rd., Forest Hills, Long Island, N. Y.

(A) POWERS, MARION BEATRICE, A.B. (Iowa) 1928, A.M. (ibid) 1929. Asst. in Speech Cor., Madison Public Schools. 1 Langdon St., Madison, Wis.

(F) PRESSMAN, JOEL JAY, M.D. (Harvard) 1925, Sc.M. (Univ. Penn.) 1931. Chief of Bronchoscopic Service, Cedars of Lebanon Hosp. 1917 Wilshire Blvd., Los Angeles, Calif.

(A) RANDALL, ESTHER FRANCES, S.B. (Minn.) 1936. Pub. Sch. T. of Speech Cor. 2 W. Lake St., Chisholm, Minn.

(F) RAUBICHECK, LETITIA ELWOOD (Mrs. Charles W. Raubicheck), A.B. (New Rochelle) 1914, A.M. (Columbia) 1928, Ph.D. (N. Y. Univ.) 1934. Dir. of Speech Imp. in N. Y. City Schools; Instr. in Speech Cor., Hunter Coll. and N. Y. Univ. 85 Mercer Ave., Hartsdale, N. Y.

(A) READ, LILLIAN FAIRCHILD (Mrs. Raymond P. Read). Pub. Sch. T. of Speech Cor. 2020 Nicollet Ave., Minneapolis, Minn.

(A) REED, NORMA DREFLE, (Mrs.), B.S. (Wash. Univ.) 1938. Instr. Speech Cor. and co-dir. of own school, South Drive, Jefferson Heights, New Orleans, La.

(A) REID, LOREN DUDLEY, A.B. (Grinnell) 1927, A.M. (Iowa) 1930, Ph.D. (ibid) 1932. Asst. Prof. of Speech, School of Speech, Syracuse Univ., Syracuse, N. Y.

(A) REIGART, JOHN F., A.B. (Dickinson) 1888, Ph.D. (Columbia) 1916. (Retired.) Prof. of Psychol. and Edu., T. Coll., Columbia Univ.; Prin., N. Y. City Pub. Sch. 31 Euclid Ave., Yonkers, N. Y.

(A) RICE, DELIGHT. Pub. Sch. Sup. of Speech Cor., Berkeley, Calif.

(F) ROBBINS, SAMUEL DOWSE, A.B. (Harvard) 1911, A.M. (ibid) 1919. Speech Corrector, Mass. General Hosp. and Mass. Div. of Mental Hygiene; Prof. of Psychol., Emerson Coll.; Managing Trustee, Institute for Speech Correction, Inc. 419 Boylston St., Boston, Mass.

(A) ROBERTS, MAUMEE, A.B. (Howard Coll.) 1935. Pub. Sch. T. of Speech Cor. and Lip Reading. 1024 S. 23rd St., Birmingham, Ala.

(F) ROBINSON, FREDERICK BERTRAND, A.B. (Col. of the City of N.Y.) 1904, A.M. (N.Y. Univ.) 1906, Ph.D. (ibid) 1907, LL.D. (Manhattan Coll.) 1927, LL.D. (Pittsburgh Univ.) 1930, LL.D. (Temple Univ.) 1931. President of the City College, Convent Ave. at 139th St., New York.

(A) ROE, VIVIAN IRENE, A.B. (Iowa) 1936. Sup. of Speech Cor., Traveling Speech and Hearing Clin., Indiana Univ. 255 Alpha Hall, Bloomington, Ind.

(A) ROUTH, RHESSA ALMEDIA, A.B. (Ind. State Teachers) 1935, A.M. (ibid) 1936. Dir. of Speech Clin., Kokomo Pub. Schools. P. O. Box 335, Kokomo, Indiana.

(A) ROWE, MARGARET LOUISE, A.B. (Evansville Coll.) 1932, A.M. (Northwestern) 1939. Dir. of Speech Cor. in Pub. Schools. 213 Dreier Blvd., Evansville, Indiana.

(F) RUSSELL, G. OSCAR, A.B. (B.Y.U.) 1915, A.M. (Columbia) 1918, Ph.D. (ibid) 1927. Dir. of Speech Science Laboratories, Ohio State Univ., Columbus, Ohio.

(A) RUSSELL, LELIA M., A.B. (Wash.) 1927, A.M. (T. Coll., Columbia) 1932. Pub. Sch. T. of Speech Cor. 809 South I St., Tacoma, Wash.

(A) RUTHERFORD, BERNEICE R. (Mrs. S. J. Rutherford), S.B. (Minn.) 1934, S.M. (ibid) 1937. Pub. Sch. T. of Speech Cor. 5223 Vincent Ave., S., Minneapolis, Minn.

(A) RUTHERFORD, KATHERINE ANN, A.B. (Arkansas Coll.) 1931, M.A. (Northwestern) 1940. Asst. Speech Re-edu. Dept. 829 Foster, Evanston, Illinois. 1960 Sheridan Road, Evanston, Illinois.

(A) RUTHERFORD, VIRGINIA, A.B. (Arkansas Coll.) 1935, A.M. (Northwestern) 1938. Pub. Sch. T. of Speech Re-edu. 1542 S. 77th St., W. Allis, Wis.

(A) RYAN, EARL H., A.B. (Univ. Pittsburgh) 1927, A.M. (Columbia) 1928. Instr. in Pub. Speaking at City Coll. 237 E. 20th St., New York City.

(A) SAGERT, LOUIS ALBERT, A.B. (Wartburg Coll.) 1927, A.M. (Iowa) 1931. Area Supervisor of Iowa Remedial Education Survey. 571 Jefferson St., Dubuque, Iowa.

(A) SCARABOBYD, HARTWELL EUGENE, A.B. (Univ. Redhawks) 1936, A.M. (Iowa) 1938, Ph.D. (ibid) 1939. Private Practice, 12311 Gorham Ave., Los Angeles, Calif.

(A) SCHMITZ, MARY H., S.B. (Minn.) 1937. Dir. of Pub. Sch. Speech Cor., Central School, Rochester, Minn.

(A) SCHULZ, OLIVE DOROTHY, Ph.B. (Univ. Chicago (1932). Pub. Sch. T. of Speech Corr. 7708 Constance, Chicago, Ill.

(F) SCOLTEN, ADRIAN HERMAN, A.B. (Wis.) 1927, M.D. (Washington Univ. in St. Louis) 1931. 105 E. Nineteenth St., New York, N. Y.

(A) SECORD, ARTHUR, A.B. (Western State T. Coll.) 1928, A.M. (Mich.) 1932. Dir. of Speech Clin., Univ. of Western Ont. (summer) and Univ. of Mich., Ann Arbor, Mich.

(A) SEVENANS, MADELINE MARIE, A.B. (Univ. of Southern Calif.) 1931. T. of Speech, Beverly Hills Schools and Santa Barbara State College. 1816 So. Bentley Ave., West Los Angeles, Calif.

(A) SHAW, M. OCLO MILLER (Mrs. Everett W. Shaw), S.B. (Mo.) 1919, A.M. (Iowa) 1929. 278 Pine St. Holyoke, Mass.

(A) SHERMAN, DOROTHY HELEN, A.B. (Iowa) 1931, A.M. (ibid) 1938. Pub. Sch. T. of Speech and English. 2805 High St., Des Moines, Iowa.

(A) SHOHARA, HELEN HIDE, A.B. (Mich.) 1926, A.M. (ibid) 1927, Ph.D. (ibid) 1932. Instructor of Phonetics, Dept. of Speech and Instructor of Human Adjustment (Speech Clinic), University of Michigan, Ann Arbor, Mich.

(A) SHOVER, JAYNE, A.B. (Iowa) 1932, A.M. (ibid) 1936. Pub. Sch. Dir. of Speech Cor. and Lip Reading Instruction. 4725 Northcote, East Chicago, Ind. Supervising Clinician (summer session) Purdue Univ.

(A) SILVERMAN, S. RICHARD, A.B. (Cornell) 1933, S.M. (Washington Univ.) 1938. Instr., T. Training Coll., Central Inst. for the Deaf. 818 S. Kingshighway, St. Louis, Mo.

(F) SIMON, CLARENCE T., A.B. (Wittenberg) 1919, A.M. (Northwestern) 1922, Ph.D. (Iowa) 1925. Dir. of Speech Clin. and Prof. of Speech Re-educ., Asst. Dean, School of Speech, Northwestern Univ., Evanston, Ill.

(A) SIMONDS, ZULMA LUNT (Mrs.). T. Speech Cor., Arlington Public Schools. 48 Bartlett Ave., Arlington, Mass.

(A) SKALBECK, OLIVER M., A.B. (St. Olaf Coll.) 1926, A.M. (Iowa) 1938. Dir. of Speech Clin. and Instr. in Speech, S. E. Mo. State T. Coll. Cheney Hall, Cape Girardeau, Mo.

(A) SKERRETT, ESTHER CURTIS, S.B. (Wayne Univ.) 1936. Pub. Sch. T. of Speech Imp. 1250 Hubbard, Detroit, Mich.

(A) SLATTENGREN, HATTIE J. E., A.B. (Gustavus Adolphus) 1929. Pub. Sch. T. of Speech Cor., Jr. High Sch., Albert Lea, Minn.

(A) SMITH, BEATRICE L. Dir. of Speech Clin., Newington Home for Crippled Children, Newington, Conn.

(A) SMITH, GEORGE-WILLIAM, A.B. (Hamline) 1920, A.M. (Northwestern) 1938. Dir. of Speech Clin., Dir. Speech and Dramatics, Russell Sage College. 171 Second St., Troy, N. Y.

(A) SMITH, NORMA R., B.A. (Brooklyn Coll.) 1938, M.A. (ibid) 1940. T. of Speech, Educational Alliance; Edu. Dir. Jewish Hospital Speech Clinic. 4418 17th Ave., Brooklyn, N. Y.

(A) SNIDECOR, JOHN C., A.B. (Univ. Calif.) 1931, A.M. (Iowa) 1937, Ph.D. (Iowa) 1940. Santa Barbara State Coll., Santa Barbara, Calif.

(F) SOLOMON, MEYER, M.D. (Cornell) 1909. Assoc. in Nervous and Mental Diseases, Northwestern Univ. Med. Sch.; Neuropsychiatrist to Mt. Sinai, Edgewater, Englewood and Woodlawn Hospitals. 185 N. Wabash Ave., Chicago, Ill.

(A) SONKIN, ROBERT, S.B. (C. C. N. Y.) 1928, A.M. (Columbia) 1929. Tutor in Public Speaking, City College, Convent Ave. at 139th St., New York City.

(A) SPECTOR, SOPHIA, A.B. (Hunter Coll.) 1931, A.M. (Columbia) 1938. T. of Speech Cor., Elizabeth Public Schools. 716 Garden St., Elizabeth, N. J.

(A) SPRIESTERSBACH, D. CARYL, B.E. (Winona State Teachers Coll.) 1939, M.A. (Iowa) 1940. Speech Asst., University of Iowa. Speech Clinic, Univ. of Iowa, Iowa City, Iowa.

(A) STEELE, EDITH LORRAINE, A.B. (Mich.) 1937, A.M. (ibid) 1939. T. of Speech and Speech Cor., Riley High Sch. 2105 S. Michigan St., South Bend, Ind.

(F) STEER, MACK DAVIS, S.B. (L. I. Univ.) 1930, A.M. (Iowa) 1933, Ph.D. (ibid) 1938. Dir. of Speech Clin. and Assoc. Prof. of Speech, Speech Clinic, Purdue Univ., Lafayette, Ind.

(A) STEINBERG, IDA. Bailey Hall, Private School for Retarded Children. P. O. Box A, care Bailey Hall, Katonah, New York.

(F) STINCHFIELD, SARA (Mrs. Charles Lyle Hawk).

(F) STODDARD, CLARA BEATRICE, S.B. (Wayne) 1932. Retired Pub. Sch. Sup. of Speech Cor. 3779 Maybury Grand, Detroit, Mich.

(A) STODDARD, JANE EASTMAN, S.B. (Columbia) 1936, A.M. (ibid) 1938. Supervisor of Special Education, State Department of Education. Richmond, Virginia.

(F) STROTHER, CHARLES R., A.B. (Univ. Wash.) 1929, A.M. (ibid) 1932, Ph.D. (Iowa) 1935. Assoc. Prof. of Speech and Psychology, Psychological and Speech Clin., Univ. of Iowa, Iowa City, Iowa.

(A) STUTENROTH, RUTH IRENE (Mrs. Ruth S. Hyde).

(A) SULLIVAN, E. MARGARET, S.B.(Minn.) 1932. Pub. Sch. T. of Speech Cor. 3220 Fremont Ave., S., Minneapolis, Minn.

(A) SULLIVAN, ELLA MARY, S.B. (Minn.) 1932. Pub. Sch. T. of Speech Cor. 3220 Fremont Ave., S., Minneapolis, Minn.

(A) SULLIVAN, HELEN BRITTON (Mrs. Paul D. Knight).

(A) SULLIVAN, MARGARET J., A.B. (Loretto Hts.) 1924, A.M. (T. Coll., Columbia) 1932. Pub. Sch. T. of Speech Cor. 2020 Nicollet Ave., Minneapolis, Minn.

(A) SWEENEY, ALMA KATHRYN (Mrs.), B.A. (Iowa) 1939. Private Practice. 615 Sunset Lane, East Lansing, Mich.

(A) TAPLIN, IDA. Sup. Speech Imp., Bd. of Educ., City Hall Annex, Newark, N. J.

(A) TAYLOR, GLENN JAMES, B.Ed. (Ill. Nor.) 1935, A.B. (Ill. Nor.) 1936, A.M. (Iowa) 1937. Asst. Prof. of Speech, Westminster Coll. 210 N. Mercer, New Wilmington, Pa.

(F) TAYLOR, JANE BLISS, S.B. (Columbia) 1920, A.M. (ibid) 1937. Chairman Speech Cor. Clin. in Bronx Buildings. Hunter Coll. 251 W. 97th St., New York City.

(A) TEMPLIN, MILDRED, A.B. (Wis.) 1936, A.M. (ibid) 1937. 106 Morningside Drive, New York City, N. Y.

(F) THOMAS, CHARLES KENNETH, A.B. (Cornell) 1922, A.M. (ibid) 1924, Ph.D. (ibid) 1930. Dir. of Speech Clin. and Assoc. Prof. of Public Speaking, Cornell. Goldwin Smith Hall, Ithaca, N. Y.

(A) THOMAS, RUTH HAZEL, Ph.B. (Syracuse) 1911, A.M. (Columbia) 1933. Dir. of Speech Clinics in Passaic Pub. Schools. 311 Mt. Prospect Ave., Newark, N. J.

(A) TIBBITS, FRANCES BEERS, S.B. (N. Y. Univ.) 1930, A.M. (Columbia) 1933. Pub. Sch. Speech Correctionist. 311 Mt. Prospect Ave., Newark, N. J.

(F) TRAVIS, LEE EDWARD, A.B. (Iowa) 1922, A.M. (ibid) 1923, Ph.D. (ibid) 1924. Dir. of Psychol. Clin. and Prof. of Psychol. and Speech, Univ. of S. Calif., Los Angeles, Calif.

(A) TURNER, MARTHA PRENTICE. Speech Correctionist in office of Dr. Walter O. Klingman, New York. 140 East 63rd St., New York, N. Y.

(A) TUTHILL, CURTIS E., A.B. (Macalester) 1935, A.M. (Iowa) 1936, Ph.D. (ibid) 1939. Dir. of Speech Clin., Temple Univ., Philadelphia, Pa.

(F) TUTHILL, DOROTHY DAVIS, A.B. (Mich.) 1933, A.M. (Iowa) 1936, Ph.D. (ibid) 1939. Speech Dept., Brooklyn College, Brooklyn, N. Y. 1931 N. 13th, Philadelphia, Pa.

(F) TWITMYER, EDWIN BURKET, Ph.B. (Lafayette) 1896, M.S. (ibid) 1898, Ph.D. (Univ. Penn.) 1902, LL.D. (Lafayette) 1933. Prof. of Psychology, Univ. of Pennsylvania. Secane, Pa.

(A) UTLEY, JEAN L., S.B. (Wash. Univ.) 1935, A.M. (Wayne Univ.) 1938. T. at N. J. School for the Deaf, W. Trenton, N. J.

(A) VAN DUSEN, CLARENCE RAYMOND, A.B. (Indiana Univ.) 1931, A.M. (Mich.) 1932, Sc.D. (ibid) 1937. Dir. of Speech Clin. and Instr. in Speech, Mich. State Coll., E. Lansing, Mich. 1415 W. Franklin St., Elkhart, Ind.

(F) VAN RIPER, CHARLES, A.B. (Mich.) 1926, A.M. (ibid) 1929, Ph.D. (Iowa) 1934. Dir. of Speech Clin., Western State Coll. 203 Fairfax, Kalamazoo, Mich.

(A) VANCE, CAROLYN, A.B. (Emerson) 1920, A.M. (Northwestern) 1940. Dir. of Speech Clin. and Asst. Prof. of Speech, Univ. of Georgia, Athens, Ga.

(A) VERGARA, ALLYS DWYER (Mrs.), A.B. (Coll. of New Rochelle) 1924, A.M. (Columbia) 1926. Dir. of Speech Clin. and Assoc. Prof. of Speech, College of New Rochelle, N. Y. 36 Laurel Place, New Rochelle, N. Y.

(A) VIAN, KATHERINE MARIE, A.B. (Western State T. Coll.) 1938. Pub. Sch. T. of Speech Cor. 401 Houston Ave., Muskegon, Mich.

(A) VOELKER, CHARLES H., A.B. (Ohio State) 1931, A.M. (ibid) 1933. Dir. of Speech Clin., 200 Old Biology Bldg., Oklahoma A. & M. Coll. 160 King's Highway, R. R. 3, Stillwater, Okla.

(A) VOGT, JUANA ALLRAUM (Mrs.), A.B. (Univ. Calif.) 1923, A.M. (T. Coll., Columbia) 1939. Carilla 2147, Lima, Peru, So. America.

(A) WADDLE, ELSIE LENORA, A.B. (Southwestern Coll.) 1920, A.M. (Iowa) 1934. Pub. Sch. T. of Speech Cor. 1648 S. Baltimore Ave., Tulsa, Okla.

(A) WALTON, EMILY. Teacher of Speech Cor. and Remedial Reading. Private Tutoring in Schools and Hospitals. 45 Avalon Rd., Milton, Mass.

(F) WARD, LAVILLA AMELIA. State Sup. of Speech Cor., State Dept. of Public Instruction, Madison, Wis.

(A) WEIDIG, PHYLLIS D., S.B. (Hunter Coll.) 1938. Special Teacher of Speech Improvement, El. and Jr. High Schools of N. Y. C. 2665 E. 27th St., Brooklyn, N. Y.

(A) WEINER, RUTH HOMMEL (Mrs.), S.B. (Northwestern) 1932. Schenley Apts., Pittsburgh, Pa.

(F) WELLS, EARL WILLIAM, A.B. (Iowa) 1921, A.M. (Wis.) 1927, J.D. (Iowa) 1928. Dir. of Speech Clin. and Prof. of Speech, Oregon State Coll. 633 N. 12th St., Corvallis, Ore.

(F) WEST, ROBERT WILLIAM, Ph.D. (Wis) 1925. Dir. of Speech Clin. and Prof. of Speech Pathol., Univ. of Wisconsin, Madison, Wis.

(A) WESTLAKE, HAROLD, A.B. (Mich. State Normal Coll.) 1928, S.M. (Mich.) 1931, Ph.D. (ibid) 1938. Asst. Prof. of Speech Cor. and Audition, Penn. State Coll. 209 W. Foster, State College, Pa.

(A) WHITTAKER, RUTH D. (Mrs. Clarence W. Bergquist).

(A) WHITTEN, IDA E., A.B. (Nebraska) 1928, A.M. (ibid) 1934. Speech Correction Dept., Pub. Schools, Racine, Wis.

(A) WICKMAN, NESTOR ALFONSE, A.B. (Mich.) 1936, S.M. (ibid) 1937. Research Asst. in Speech Cor. and Audiometry, Univ. of Mich. 203 N. Ingalls, Ann Arbor, Mich.

(F) WILE, IRA S., A.B., B.S. (Rochester) 1898, M.S. (ibid) 1908, M.D. (Univ. Penn.) 1902. Dir. of Speech Clin. of Health Class at Mt. Sinai Hosp.; Lecturer on Disorders of Conduct and Personality, Columbia Univ., Brooklyn Coll., and Hunter Coll. 264 W. 73rd St., New York City.

(A) WILKE, WALTER H., A.B. (Wis.) 1928, A.M. (ibid) 1929, Ph.D. (Columbia) 1934. Asst. Prof. of Speech, N. Y. Univ., Washington Sq., East, New York City.

(A) WILKIE, LILLIAN R., S.B. (Wayne) 1936, Pub. Sch. T. of Speech Imp. 3311 Gladstone Ave., Detroit, Mich.

(A) WILKINSON, M. JUSTUS, A.B. (Univ. Denver) 1934, A.M. (ibid) 1938. Instr. of English and Speech, Dept. of English and Speech, Iowa State Coll., Ames, Iowa.

(A) WILLIAMS, GRACE M., A.M. (Northwestern) 1935. Dir. of Speech Clin. and Instr. in Speech, E. Ill. State T. Coll. 819 Jackson St., Apr. 2, Charleston, Ill.

(A) WILLSEA, MARY A., A.B. (Denver) 1924. Pub. Sch. T. of Speech Cor. 4669 Quitman St., Denver, Colo.

(A) WILSON, MARGARET BOYD (Mrs.), A.B. (Emerson) 1937. N. H. Mental Hygiene Clin., Dept. of Psychology. 105 Pleasant St., Concord, N. H.

(A) WOKURKA, ROSE ANNE, A.B. (Coll. of St. Catherine) 1932, A.M. (Minn.) 1934. Instructor in Speech Correction, Mary Manse College, Toledo, Ohio.

(A) WOOD, ALICE LOUISA. Special Teacher of Speech Improvement, New York City. 8786 153rd St., Jamaica, N. Y.

(A) WOOD, ALICE MORTON, B.S. in Ed. (Boston Univ.) 1930. T. of Speech Cor. and Asst. in the Clinics, Univ. of Boston. 88 Harvard St., Newtonville, Mass.

(F) WOOD, HARRY THOMAS, A.B. (Ill.) 1915, A.M. (Wis.) 1923. Dir. of Speech Clin. and Asst. Prof. of Public Speaking, Mich. State Nor. Coll. 938 W. Forest Ave., Ypsilanti, Mich.

(A) WOOTTON, MARY HOOPER (Mrs. Richard Lambert Masland).

(A) WRIGHT, JULIA M., A.B. (Denver) 1928. Pub. Sch. T. of Speech Cor. 4669 Quitman St., Denver, Colo.

(A) YOAKAM, DORIS G., A.B. (S. Calif.) 1931, A.M. (ibid) 1932, Ph.D. (ibid) 1935. Dir. of Speech Clin., and Prof. of Speech, N. Ill. State T. Coll., De Kalb, Ill.

(A) YOUNG, ADELBERT JACOB, A.B. (Wis.) 1924. Dir. of Pub. Sch. Speech Clin. 638 State St., Oconomowoc, Wis.

(A) YOUNG, EDNA HILL. Dir. of Speech Clin., Hill-Young School, 2716 Ellendale Pl., Los Angeles, Calif.

(A) ZEILE, ADELAIDE, A.B. (Western Reserve Univ.) 1920, A.M. (Wis.) 1930. Pub. Sch. T. of Speech. 11508 Mayfield Rd., Cleveland, Ohio.

(A) ZEITLIN, ROSALYN ROSENTHAL (Mrs. N. S. Zeitlin), A.B. (Wis.) 1930, A.M. (Northwestern) 1936. Dir. of Speech Clin., Michael Reese Hosp. 708 Buena Ave., Chicago, Ill.

(F) ZERLER, MARY LOUISE, S.B. (T. Coll., Columbia) 1931, A.M. (ibid) 1936. Pub. Sch. Dir. of Speech Cor., Yonkers, N. Y. 517 W. 113th St., New York City.

(A) ZIMMER, CATHERINE H., A.B. (Mich.) 1932, A.M. (Northwestern) 1939. T. of Speech Cor., Shorewood Pub. Schools. 4305 N. Marlborough Dr., Milwaukee, Wis.

(F) ZIMMERMAN, JANE DORSEY (Mrs. Max Zimmerman), S.B. (Columbia) 1922, A.M. (ibid) 1923. Assoc. in Speech, T. Coll., Columbia Univ. 501 W. 120th St., New York City.

NECROLOGY

HENRIETTA PRENTISS

GEOGRAPHICAL DIRECTORY

ALABAMA: *Birmingham*—Roberts, M.; Birmington-Southern College—Evans, M. F.

ARIZONA: *Tucson*—University of Arizona—Cable, W. A., Huyck, E. M.

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GEORGIA: *Athens*—University of Georgia—Vance, C.; *Atlanta*—Davison, L. D.; *Statesboro*—Georgia State Teachers College—Jones, M. J.

FLORIDA: *Miami*—University of Miami—Meyer, M. F.; *Miami Beach*—Moore, R.; *St. Petersburg*—Howe, A. S.

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1927 1928	Cincinnati Chicago	West	Stinchfield	Travis, Borden
1929 1930	New York City Chicago	Kenyon	Stinchfield	Robbins, Busse
1931 1932	Detroit St. Louis	Kenyon	Robbins	Gifford, Bryngelson Blanton, West Ward, Goldstein
1933 1934	New York City Chicago	Blanton	Robbins	Travis, Bryngelson Hawk, Kenyon Goldstein, Gifford
1935 1936	Chicago St. Louis	Travis	Robbins	Solomon, Bryngelson Hawk, Blanton Bender, Gifford
1937 1938	Chicago Cleveland	Goldstein	Robbins	Hawk, Bryngelson Russell, Travis Bender, Bluemel
1939 1940	Chicago State College, Pa.	Hawk	Robbins	Hawk, Bryngelson, Russell, Goldstein, Simon, Kantner

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